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SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

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SURGERY

VOL. 35 JUNE, 1954 NO. 6

Original Communications

SIMPLIFICATION OF THE BILLROTH I GASTRIC RESECTION

KOMEI NAKAYAMA, M.D.,* CHIBA, JAPAN

FOREWORD

ALTHOUGH various operative methods have been employed to treat duodenal ulcer radically, the ulcer is not usually removed by techniques commonly employed. This is due to the surgical problems of dissection in the location of the ulcer with a consequent increased mortality. Hence, gastrectomy without removal of the ulcer, or gastroenterostomy, has been the common operative management of duodenal ulcer in the past.

The development of a proper operative method preventing the recurrence of preoperative complaints and peptic ulcer has been the hope of surgeons for many years.

We have devised a new technique of excision of the duodenal ulcer with a low operative mortality and recurrence rate.

OPERATIVE METHOD

The procedure is performed using spinal anesthesia through an upper midline abdominal incision. First, the greater curvature of the stomach is cleared by ligating the gastrocolic ligament in three or four places. The axis of the right gastroepiploic artery and Mayo's vein are ligated. Almost all duodenal ulcers are located in the duodenal cap, and frequently cause adhesion formation to surrounding organs. Freeing of the adherent and thinned portion of the duodenum is reserved as the final stage of resection. The uninvolved serosa is located distal to the ulcer and mobilization from surrounding tissues is proceeded with, in the direction of the gastric pylorus. The portion invaded by the degenerating ulcer process and normal tissue can be clearly differentiated.

The lesser curvature of the stomach is mobilized to the cardiac end of the stomach, and the line of resection is determined. Subtotal gastrectomy (more than two-thirds of the stomach) is preferred in our clinic. My gastric clamp is then placed at the line of resection to amputate the stomach (Fig. 1). The upper portion of the gastric remnant is closed by continuous Lembert sutures. The distal posterior wall of the gastric pouch is fixed to the head of the pancreas

Received for publication, Sept. 30, 1953.

*Professor of surgery, School of Medicine, Chiba University.

by three interrupted silk sutures (Fig. 2,*A* and *B*). Tension on the anastomosis is thereby completely eliminated. The forceps which was placed on the duodenal remnant is removed at this juncture. The ulcer is cicatrix can be further removed when necessary so that normal mucosa is available for anastomosis to the gastric remnant. When the cicatrix extends toward the posterior wall of the duodenum, special attention is required to avoid incising the pancreas.

Many cases present problems which demand a special approach. 1. When the ulcer has penetrated adjacent structures, the base of the ulcer must be excised. Almost all of the ulcer can be removed with the resected stomach, leaving the base adherent to the adjacent organ. The remaining base must be curetted and covered by normal gastric serosa at the completion of the anastomosis. 2. When stenosis is found in the descending portion of the duodenum, a longitudinal incision is made in the duodenum to enlarge the cut end, making it more suitable for anastomosis with the gastric pouch (Fig. 2, *C*).

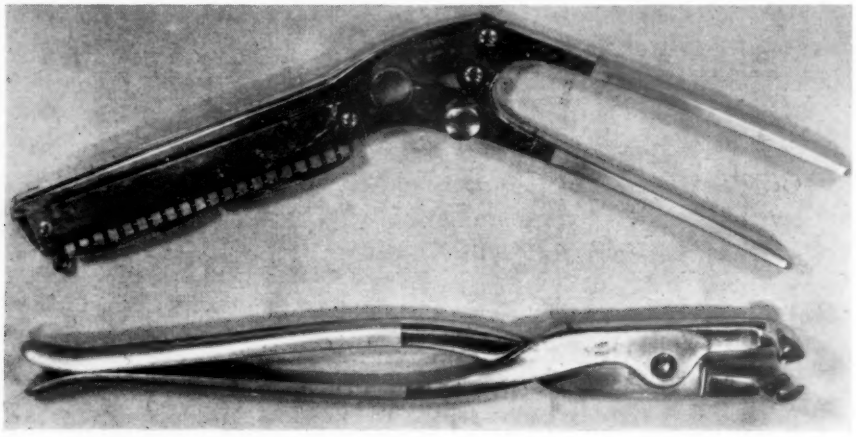


Fig. 1.—My clip placer, an instrument adapted from the V. Petz suturing apparatus. These are removal cartridges into which the clips are placed. The écraseur type of instrument shown is used to drive the clips home. The clip placer has a dorsal groove into which a knife blade is inserted to divide the tissues which are to be separated.

Over 90 per cent of duodenal ulcers can be excised by these means. Reestablishment of gastrointestinal continuity is made by a gastroduodenal anastomosis. The posterior wall of the anastomosis is a single continuous suture (Fig. 2, *D*). The anterior wall anastomosis is performed in two layers, using an Albert mucosal suture covered by a row of Lembert sutures. The upper and lower ends of the anastomosis have sutures which pass through the gastric serosa and pancreas to the duodenal serosa, thereby reinforcing the weakest portion of the anastomosis. The head of the pancreas plays a role as splint between the stomach and duodenum (Fig. 2, *D*). The anastomosis is drawn to the left by this operative technique, but the head of the pancreas is pulled with the duodenum, and tension of the anastomosis is avoided. The anastomosis usually lies to the left of the vertebral column. The operation is completed by covering the anastomosis with greater omentum and closing the abdominal wall.

A rubber tube (Levin tube) is left in the stomach for two or three days after the operation to prevent distention. The patient is discharged from the

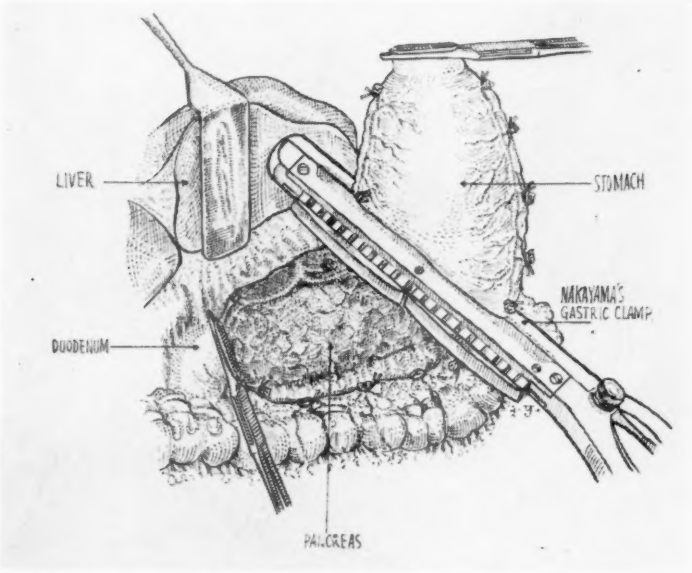
hospital on the twelfth or thirteenth postoperative day. The operative time for this procedure which requires a minimum of manipulation is usually thirty minutes.

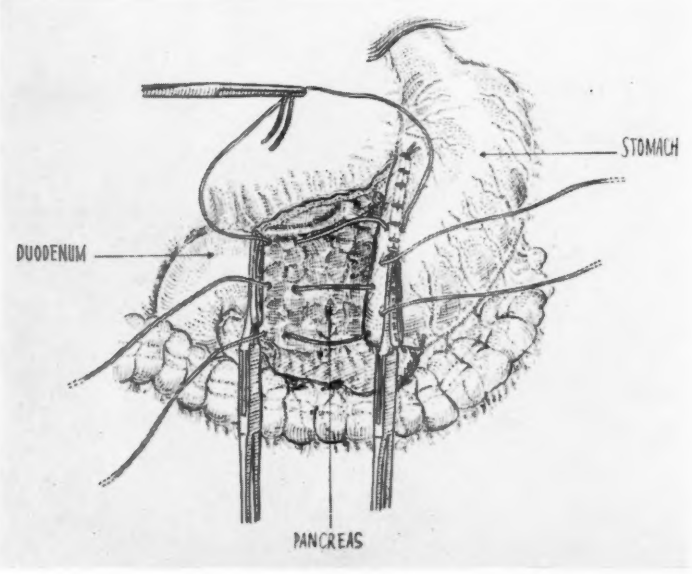
OPERATIVE RESULTS

Since August, 1946, 687 patients have been operated upon by this technique.

There have been three hospital deaths following the operation, a mortality rate of 0.4 per cent.

A.

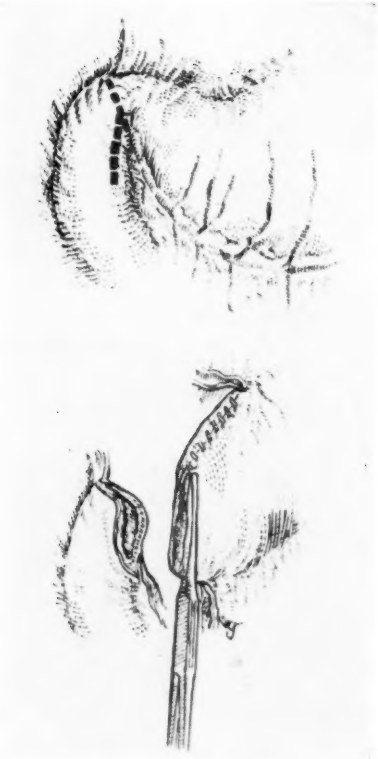


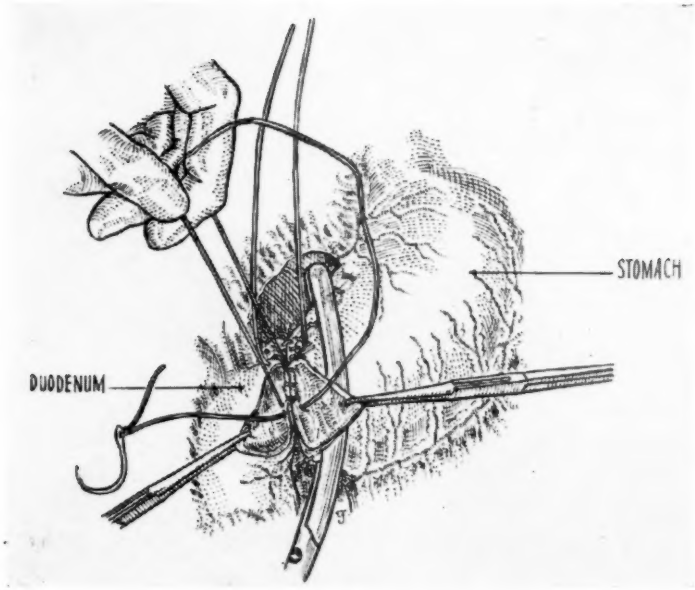


B.

Fig. 2, *A* and *B*.—My technique of gastric resection by the Billroth I method. *A*, The segment of stomach to be removed. The duodenal ulcer is excised in 90 per cent of instances. *B*, Partial closure of the lesser curvature; placement of anchoring sutures between the posterior gastric wall and the pancreatic capsule behind the duodenum.

C.



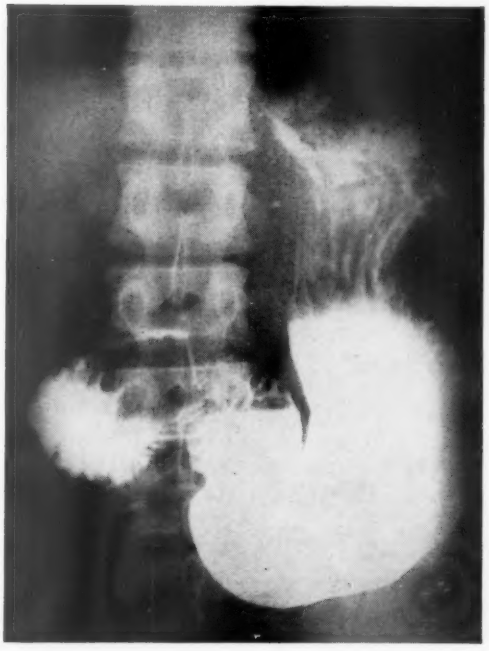


D.

Fig. 2, *C* and *D*.—*C*, Method of enlarging the duodenum for the anastomosis. *D*, A continuous suture approximates the posterior walls of stomach and duodenum. This suture is continued anteriorly, and the first stitch of the anterior wall of continuous Lembert suture has been placed previously. Posteriorly, this continuous Albert suture is employed only, for the purpose of anchoring sutures.

The proportion of duodenal ulcer to gastric ulcer in Japan is quite different from that in the United States. Of the patients mentioned, 258 had a duodenal ulcer, or only 37.6 per cent of the series of gastroduodenal ulcers. The rate of resectability of all gastroduodenal ulcers was 99.0 per cent, a higher figure than is usually seen in the larger Japanese hospitals.

A.



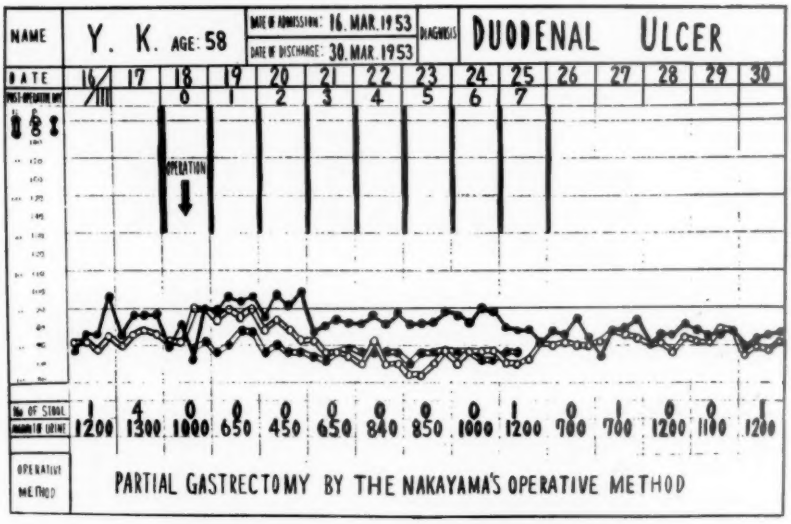
B.

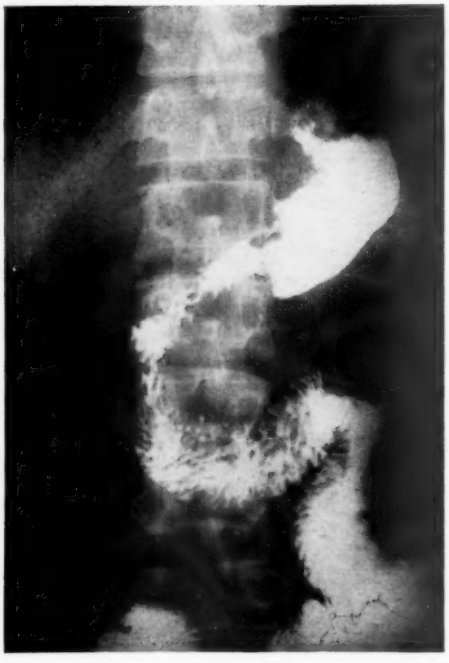
Fig. 3, *A* and *B*.—Clinical data in a resection case. *A*, Roentgenogram before operation. *B*, The excised stomach.

The pancreatic function of these patients was carefully studied, and no abnormalities were found (Fig. 2, *D*). Hepatic function has shown no changes.

In an illustrative case of duodenal ulcer managed by the previously outlined technique, preoperative roentgenograms were as illustrated in Fig. 3, *A*. The removed specimen is illustrated in Fig. 3, *B*. Fig. 3, *C* shows the postoperative course of the patient. Fig. 3, *D* illustrates the postoperative roentgenogram of the patient.

C.


NAME
Y. K. AGE: 58
DATE OF ADMISSION: 16. MAR. 1953
DATE OF DISCHARGE: 30. MAR. 1953
DIAGNOSIS
DUODENAL ULCER
DATE
16 17 18 19 20 21 22 23 24 25 26 27 28 29 30
POST-OPERATIVE DAY
0 1 2 3 4 5 6 7
OPERATION
No. OF STOOL
AMOUNT OF URINE
1200 1300 1000 650 450 650 840 850 1000 1200 700 700 1200 1100 1200
OPERATIVE METHOD
PARTIAL GASTRECTOMY BY THE NAKAYAMA'S OPERATIVE METHOD



D.

Fig. 3, *C* and *D*.—*C*, The postoperative fever chart. *D*, Postoperative roentgenogram.

CLINICAL INVESTIGATION AT THE TIME OF FOLLOW-UP

Excluding the patients who died of accidental or unrelated causes, all patients were gainfully employed in their normal occupation by six months postoperatively. Routine clinical examination is normal in most cases. No anemia was found; most patients were gaining weight at this time. No recurrent peptic ulcers or bleeding has been encountered.

Complaints after the operation have been carefully studied, and nothing of a severity to require a change or discontinuance of occupation was found. No ulcer pain was seen following the operation. Abdominal pain due to ascariasis was encountered, but disappeared on treatment of the ascariasis. Vomiting has occurred since surgery in 8.4 per cent of cases. Constant vomiting was seen in 2.8 per cent. Sixteen per cent of all patients operated on by this method have diarrhea. In most patients this was not severe (two bowel movements a day). This difficulty ameliorates with passage of time.

No objective evidence of recurrence exists in these patients, and the general condition of the patients is satisfactory.

SUMMARY

The radical surgical treatment of duodenal ulcer by this operative method, that is, Billroth I gastrectomy with the alterations in technique suggested by me, gives good results; in fact, the postoperative condition of patients subjected to this operation compared very favorably with other surgical procedures. The operative technique is quite simple. Normal alimentary continuity is re-established.

CONCLUSIONS

1. In the radical surgical treatment of the duodenal ulcer, the ulcer as the source of the complaints should be removed, together with more than two-thirds of the stomach.

2. It has been thought that subtotal gastrectomy can be performed safely by the employment of a Billroth II gastrectomy. However, this operative method prevents the removal of the duodenal ulcer with the stomach.

3. I have modified the technique of the Billroth I gastrectomy, introducing a new thought in re-establishment of the alimentary tract. We fix the posterior wall of the stomach to the head of the pancreas, moving the gastroduodenal anastomosis to the left; thus we have succeeded in removing a sufficiently large amount of stomach as well as the duodenal ulcer, something which is difficult or impossible in many Billroth II resections.

4. We have treated 687 cases of gastroduodenal ulcers by this method; 285 were duodenal ulcers, all of which were cured. We have not experienced a single case of suture insufficiency, the most dangerous factor when a direct gastro-duodenal anastomosis is performed after the subtotal gastrectomy.

5. Follow-up studies fail to disclose any recurrences.

A STAINLESS STEEL CANNULA FOR THE CREATION OF GASTROINTESTINAL FISTULAS

S. Frank Redo, M.D., and William A. Barnes, M.D., New York, N. Y.

(From the Department of Surgery, Cornell University Medical College, and the New York Hospital)

GASTRIC fistulas have been used by many investigators for the study of gastric secretion. These have been formed (1) directly as a gastrostomy,[1, 2] (2) by interposing a segment of ileum between the stomach and skin,[3] or (3) by using a cannula. Pavlov[4] described a spool-shaped cannula, the lumen of which was blocked by a cork stopper when samples were not being taken. Dragstedt and Ellis[5] used a gold-plated cannula that was brought out to the skin.

This paper describes a spool-shaped cannula that has been found to be useful in creating gastric fistulas in dogs. Its chief advantages are that it is leakproof, well tolerated by the animals, and causes no skin reaction about its point of exit.

Description.—The cannula, made of stainless steel, consists of a central hollow cylinder internally threaded, with a flange at each end. The flanges are perforated near the periphery so that they may be sutured to the stomach and skin. This helps to stabilize the cannula. However, it has been found unnecessary to suture the flange to the skin. A stainless steel screw inserts into the central hollow cylinder to prevent leakage (Fig. 1). When gastric specimens are desired, the screw is removed and samples are readily obtained.

Specifications of Cannula.—Length is 2.5 cm., diameter of flanges 3 cm., diameter of central hollow cylinder (lumen) 0.6 cm. There are eight perforations near the periphery of the flange, each with a diameter of 1 mm. and spaced 1 cm. apart. A modification of this cannula is shown in Fig. 2. This type of cannula has a single flanged end which fits into the stomach. The central hollow cylinder is threaded on the inside and on the outside. The inner threads are for the stainless steel screw. The skin flange screws over the outside of the central hollow cylinder. A second smaller nut is then screwed down over this to prevent the flange from being accidentally unscrewed. This is superior to the original model (although slightly more expensive) since it allows for variation in thickness of the abdominal wall. In addition, the opening in the skin need be only large enough to accommodate the central cylinder.

The cannula is easily inserted, as follows: An incision is made through the wall of the stomach. The cannula is inserted and the stomach closed about it by a purse-string suture or interrupted inverting sutures of nonabsorbable material. Several sutures are then placed through all layers of the stomach and through the perforations in the gastric flange. This anchors the flange within the stomach. A piece of omentum is wrapped around the central cylinder. A

Aided by a grant from the Wyeth Foundation and Mrs. Percy Strauss.
Received for publication, Oct. 6, 1953.

stab wound is made through the abdominal wall, and the free flange of the cannula is brought through this to the surface of the skin. It is not necessary to anchor this flange to the skin.

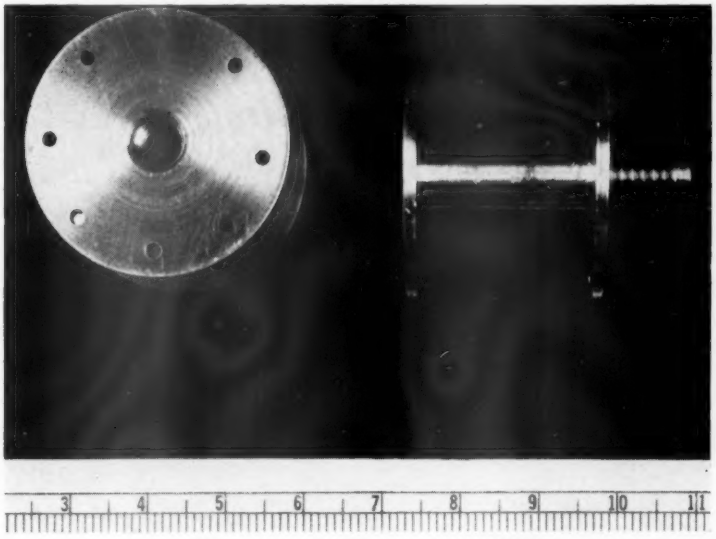


Fig. 1.—Cannula for creation of gastrointestinal fistula. Specifications are listed in the text.

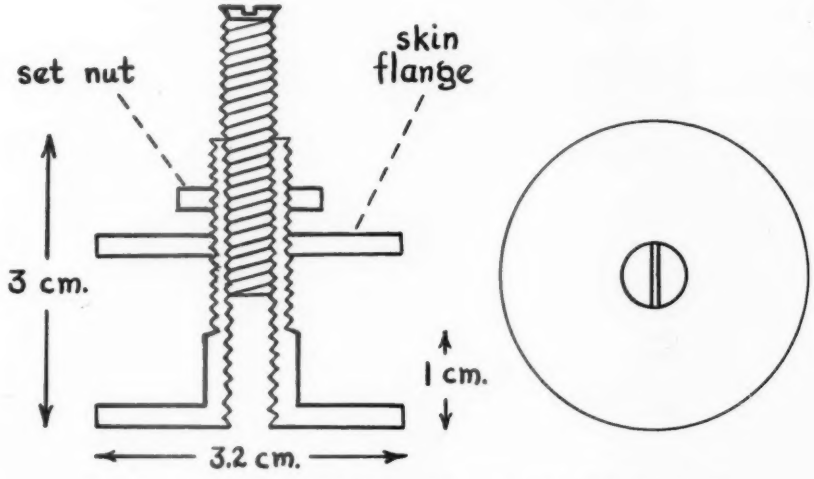
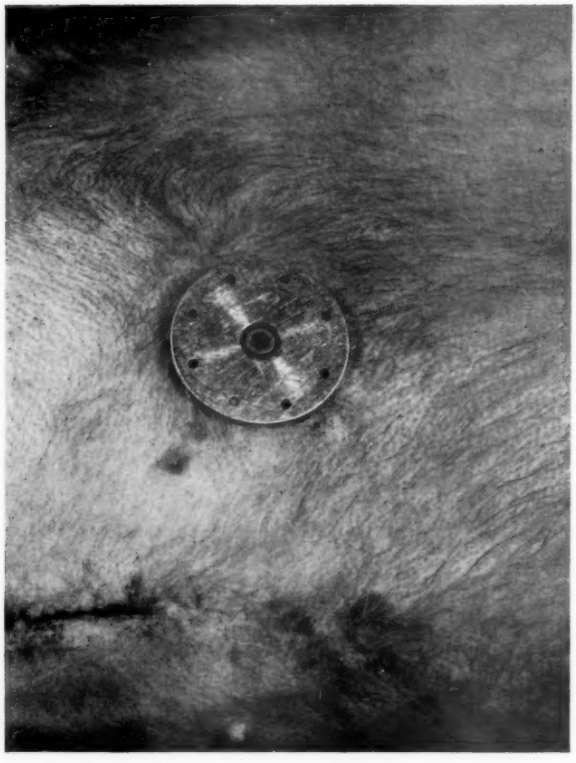


Fig. 2.—Modified cannula (cross-section and end-on views).

The cannula described has been used to create gastric fistulas in twenty dogs. There was no leakage and no maceration or infection of the skin. All animals tolerated the cannula well, several for more than six months (Fig. 3). They exhibited no interest in it and did not attempt to bite or remove it. Gastric specimens were obtained by removing the central screw and collecting the secretions in a small beaker held beneath the skin flange.

Modifications of this cannula can be used for many types of gastrointestinal fistulas. At present, work is in progress to evaluate its use in colostomy. In this connection, absence of leakage following irrigation and prevention of stricture may be among its advantages.

A.



B.

Fig. 3, *A* and *B*.—Cannula two months after insertion in the stomach of a dog. Note the absence of changes in the skin about the cannula.

SUMMARY

1. A modified spool-shaped cannula for the creation of various types of gastrointestinal fistulas is described.

2. The cannula has been used in twenty dogs and has been tolerated well. There has been no leakage or maceration or infection of the skin about the site of exit.

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RETURN OF SODIUM LOADS IN NORMAL SUBJECTS FOLLOWING RESTRICTED SODIUM INTAKE

J. U. SCHLEGEL, M.D., ROCHESTER, N. Y.

(From the University of Rochester, School of Medicine and Dentistry, and the Department of Surgery, Buswell Urological Research Laboratory)

THE prevention of sodium retention is a subject of great interest since a successful correction of this is important for the treatment of a great number of pathologic conditions. We have been studying this phenomenon, especially as it occurs following severe trauma, such as burns, and certain major operations. It was found that the increase in adrenal activity occurring in seriously burned patients does not necessarily diminish the ability to excrete sodium. In fact, it was observed that when sufficient amounts of hypertonic sodium chloride were given relatively soon following the trauma, the return of subsequent loads of sodium was higher than that found in normal persons.[1] It was also found that the urinary pH can only exhibit normal fluctuations when base excretion (sodium and potassium) is above approximately 100 microequivalents per minute, provided that titratable acidity is within a normal range of 10 to 25 microequivalents per minute.[2] The amount of hypertonic sodium chloride initially given the burned patients was, therefore, considered sufficient only when urinary pH fluctuations were maintained or restored.

Because this method of treatment is based upon the assumption that sodium retention did not exist prior to the trauma,[3] we must be very careful to rule out an unsuspected pre-existing condition of this sort. Black, Platt, and Stanbury[4] have shown that normal persons who have been on a low-sodium diet for five days would retain sodium when subjected to a load of intravenously administered 5 per cent sodium chloride. This observation seems to suggest that patients preoperatively occasionally might exhibit sodium retention, since their intake often may be low, or an actual loss might have been encountered by vomiting, diarrhea, or other routes. Since surgery in itself will lead to sodium retention, a pre-existing condition of this sort may even be exaggerated by the operation. This study is an attempt to evaluate the time necessary for establishing a sodium retention.

EXPERIMENTAL PROCEDURES AND METHODS

Twenty-one male medical students were used. Eleven of these received a low-sodium diet containing approximately ½ Gm. of sodium chloride and 3 Gm. of potassium chloride daily. The daily sodium intake in milliequivalents for each student represented in alphabetic order is given in Table I. The first

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The excellent technical assistance of Mr. William R. Johnson, Miss Patricia Bryant, and Miss Sally Fox is gratefully acknowledged.

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five (A, B, C, D, and E) received, in addition, 3 Gm. of sodium chloride (a total of 60 meq.) on the first day of the experiment.

The return of an oral sodium load was in all cases calculated as the percentage return over a twenty-four hour period (8:00 A.M. to 8:00 A.M.) of the total amount of sodium given during the daytime of that period. A water load was given to several of the students. This is indicated in Table I by +. All the water loads given to these as well as other of the students amounted to 800 c.c. per square meter of surface area. The return was then calculated on a four-hour basis, and most of the patients had their weights followed prior to and after the water load. Since a sodium load of a magnitude given to the first five students did not exceed the amount of sodium they had lost during that period (Fig. 1), two students, F and G, were put on a low-sodium diet for eight days and were then given 15 Gm. of sodium chloride over one day. One of these (F) was given a second sodium load of the same magnitude the next day. The same procedure, except for the sodium load, was followed for two other students (H and I), who received a water load on the ninth day of their low sodium intake. J is a control study where a normal individual was subjected to a low sodium intake for eleven days. K was also subjected to a low-sodium diet, and received on the ninth day an acid load in the form of 15 Gm. of ammonium chloride. This was done to try to simulate the effect of a sudden acid load on an individual who had been on a low-sodium diet, and was thought to be somewhat comparable to the situation which may arise when a patient who has been on a low sodium intake is subjected to a sudden stress. This individual received a water load on the eleventh day.

The low-sodium diet is considerably less acid than most normal diets. It was found in all the students on this diet that the amount of titratable acid they excreted was greatly diminished.

To try to simulate the conditions of a patient preoperatively, where some degree of acidosis might be present, six students were given a low-sodium diet and, in addition, a varying amount of ammonium chloride daily. Three of these were subjected to a water load and three to an oral salt load. Since, however, the loss of sodium was very extensive in these individuals, and since for practical reasons not much more than an additional amount of 15 Gm. of sodium chloride could be given daily, they were given identical consecutive loads on the two following days, as can be seen in Table I (O, P, and Q). Also in Table I can be seen the exact amount of ammonium chloride (in grams) that was given daily. It will be noted here that some received an increasing amount of ammonium chloride, while two received an amount which did not change after the fourth day.

To evaluate the effect of starvation, four students were finally subjected to complete starvation from three to four days. Two of these received a water load and two an oral sodium load at the end of the starvation.

Serum sodium, potassium, chloride, and Co_2 were determined in all students prior to the experiment and on the last day. The group which received am-

TABLE I. EXPERIMENTAL PROCEDURE*

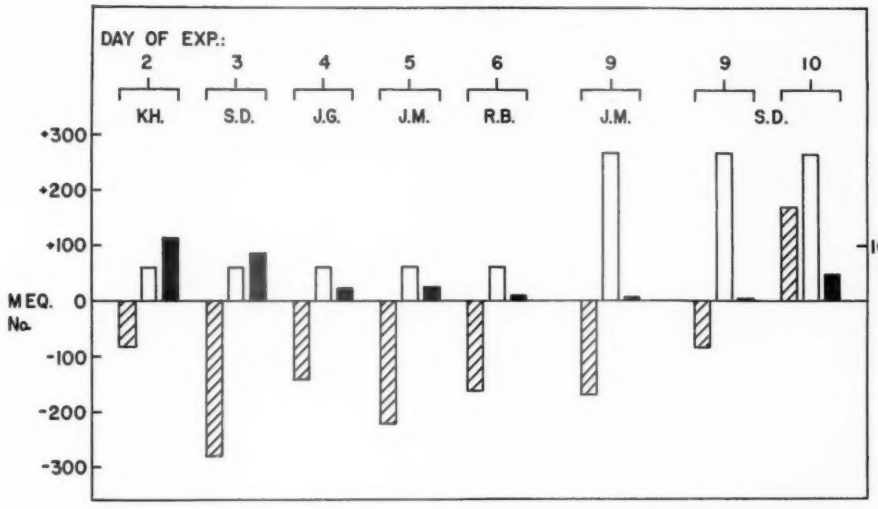
DAY OF EXPERIMENT		LOW SODIUM DIET ONLY											LOW SODIUM DIET AND AMMONIUM CHLORIDE						STARVATION			
		A K. H.	B S. D.	C J. G.	D J. M.	E R. B.	F S. D.	G J. MC.	H P. M.	I K. H.	J J. C.	K R. H.	L R. P.	M J. M.	N R. E.	O W. L.	P W. C.	Q R. M.	R P. M.	S R. M.	T W. C.	U J. B.
	Na	60	60	60	60	60	9	9	9	9	9	9	9	9	9	9	9	9	0	0	0	0
1	NH_4Cl												15	15	15	15	15	15				
	H_2O																					
	Na	60	9	9	9	9	9	9	9	9	9	9	9	9	9	9	9	9	0	0	0	0
2	NH_4Cl												15	15	15	15	15	15				
	H_2O		+																			
	Na		60	9	9	9	9	9	9	9	9	9	9	9	9	9	9	9	0	0	265	0
3	NH_4Cl												15	15	15	15	15	15				
	H_2O			+															+			
	Na			60	9	9	9	9	9	9	9	9	9	9	9	9	9	9		0		265
4	NH_4Cl												10	10	10	10	10	10				
	H_2O				+															+		
	Na				60	9	9	9	9	9	9	9	9	9	9	9	9	9				
5	NH_4Cl												10	10	10	10	10	10				
	H_2O					+																
	Na					60	9	9	9	9	9	9	9	9	9	265	265	265				
6	NH_4Cl												10	15	15	15	15	10				
	H_2O												+	+	+							
	Na						9	9	9	9	9	9		9	9	265	265	265				
7	NH_4Cl													20	20	20	20	10				
	H_2O																					
	Na						9	9	9	9	9	9		9	9	265	265	265				
8	NH_4Cl													20	20	20	20	10				
	H_2O													+	+							
	Na						265	265	9	9	9	9										
9	NH_4Cl											15										
	H_2O								+	+												
	Na						265	9	9	9	9	9										
10	NH_4Cl																					
	H_2O																					
	Na										9	9										
11	NH_4Cl																					
	H_2O											+										

*For each student is given the daily intake of sodium expressed in milliequivalents. In the group (L to Q) which received ammonium chloride, the daily intake is expressed in grams. The students who received a water load can be found in the table where + indicates the day in the experiment on which the water load was given.

monium chloride and the group which was subjected to starvation had blood chemistry determined daily. In addition, blood chemistry was determined prior to and following sodium loads as well as water loads.

The day prior to the experiment, every day during the experiment, and one day following, the urine was collected hourly from 8:00 in the morning until

Fig. 1.



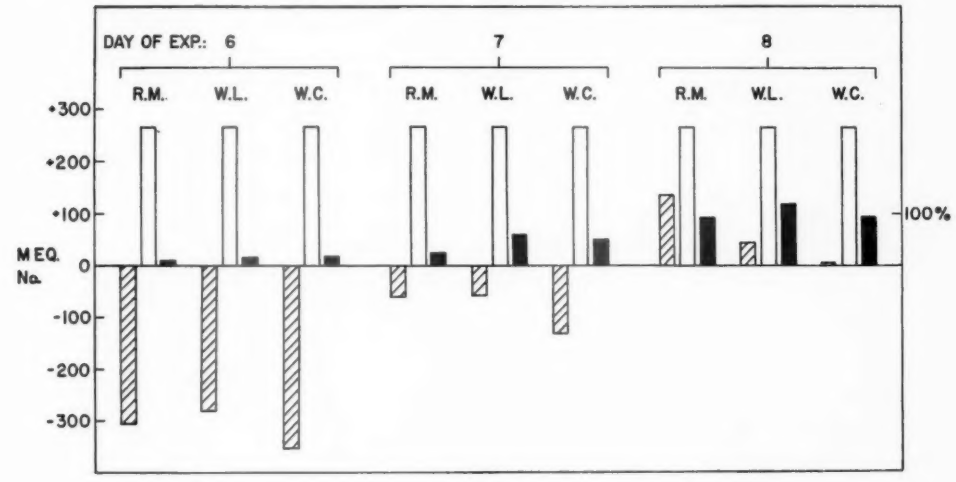


Fig. 2.

Fig. 1.—Crosshatched column: Cumulative urine sodium excretion in excess of intake from beginning of experiment until time of sodium load (milliequivalents). White column: Sodium load (milliequivalents). Black column: Return of sodium following sodium load (per cent in twenty-four hours).

The students included in this figure were on a low-sodium diet for a varying number of days. It will be noticed that the percentage return of a sodium load is lower the longer the low sodium intake has lasted. This drop in percentage return (black column) is apparently independent of the total amount of sodium lost (crosshatched column) and of the size of the sodium load (white column).

Fig. 2.—Crosshatched column: Cumulative urine sodium excretion in excess of intake from beginning of experiment until time of sodium load (milliequivalents). White column: Sodium load (milliequivalents). Black column: Return of sodium following sodium load (per cent in twenty-four hours).

The three students presented in this figure received, in addition to a low-sodium diet, varying amounts of ammonium chloride (see text for details). It will be noticed that the return of a sodium load (white column) did not reach 100 per cent before the third consecutive day of sodium loading.

4:00 in the afternoon. On these hourly collected urine samples, the pH, the specific gravity, and the volume were determined. The hourly samples were then pooled and analyzed for sodium, potassium, ammonia, titratable acidity, and chloride. The urine voided from 4:00 in the afternoon until 8:00 the next morning was also pooled and subjected to the same analyses.

The students were weighed prior to the experiment and every day during the experiment at 8:00 A.M. and 4:00 P.M.

All the experiments were carried out during winter and spring, and a routine of physical activity was established to minimize extrarenal sodium loss by sweating. The methods used for the determination of the different electrolytes in serum as well as urine have been dealt with in earlier publications, to which the reader is referred.[2]

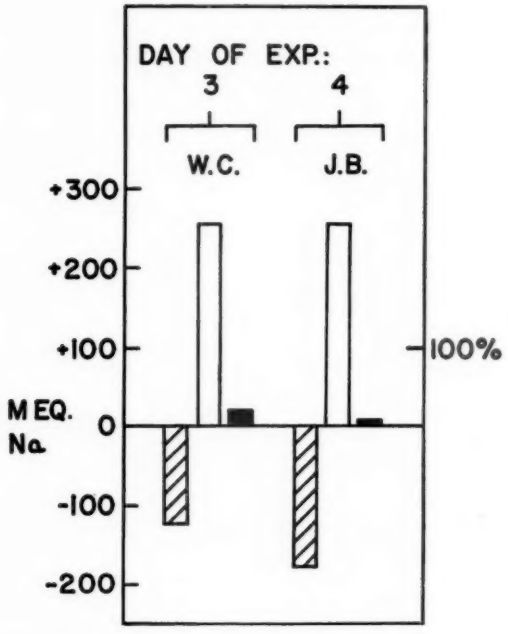


Fig. 3.—Crosshatched column: Cumulative urine sodium excretion in excess of intake from beginning of experiment until time of sodium load (milliequivalents). White column: Sodium load (milliequivalents). Black column: Return of sodium following sodium load (per cent in twenty-four hours).

The two students presented in this figure were on the third and fourth day of starvation, respectively, when they received an oral sodium load (white column). The percentage return (black column) in both cases is down, but again the one who has been deprived of sodium the longest has the lowest return following a sodium load.

RESULTS

Some of the results of sodium loading are seen in Figs. 1, 2, and 3. For each individual is shown the total urinary loss of sodium in excess of the intake in milliequivalents from the beginning of the experiment until the time the sodium load was started (crosshatched column). The negativity of the sodium balance prior to the sodium load, however, is not known exactly, since the amount of sodium lost by other routes than through the urine was not determined. Consequently, the crosshatched column can only be the smallest negative balance

possible. Since, however, the daily excretion on the low-sodium diet was very close to the intake, we can assume that the amount lost by other routes was negligible.

The white column indicates the size of the sodium load in milliequivalents and the black column shows a twenty-four hour return of this load in percentage.

In Fig. 1 are represented the students on known and low-sodium diet (A to G in Table I) who were given an oral sodium load. It can readily be seen that the percentage return (black column) drops gradually in accordance with the length of time the subjects received a low sodium intake. The percentage return seems completely independent of the total sodium loss in the urine (cross-hatched column) and of the magnitude of the load (white column).

Subject S. D. (F in Table I), who received a sodium load on the ninth day of the experiment, was subjected to a second load on the tenth day. Since on the ninth day he retained practically all 265 meq. of sodium given, he probably was in positive balance on the tenth day. On this day, the percentage return was considerably higher, but he still retained more than one-half the amount given and thus increased his retention. His weight increased 600 grams on the ninth and another 600 grams on the tenth day. Serum sodium, potassium, chloride, and CO_2 did not change significantly during the entire experiment in any of these individuals.

The effect of an oral sodium load on three consecutive days given individuals who have received a low-soduim diet as well as ammonium chloride is depicted in Fig. 2. It will be seen in Table I that the subjects found under the letters O (W. L.), P (W. C.), and Q (R. M.) received, during the time they were subjected to the sodium load, ammonium chloride in different dosages. Subjects W. L. and W. C. received increasing amounts of ammonium chloride, while subject R. M. was unchanged on a 10 Gm. daily intake after the fifth day. The total amount of sodium lost in the urine in excess of the intake is considerably higher than that which is found in the individuals who were on a low-sodium diet only. The amount of potassium lost was equally high and also much greater than that found in the persons who received a low-sodium diet only. The percentage return in the student who received the lowest dosage of ammonium chloride is somewhat of the same magnitude as the one found in the subject receiving a low-sodium diet only for the same number of days. However, the weight increase was considerably higher, amounting to 1,100 Gm. The two students receiving an increasing amount of ammonium chloride both showed a slightly higher return of the sodium load; the weight increase for Subject W. L., however, also amounted to 1,100 Gm., while Subject W. C. showed a weight gain of only 400 Gm. It will be noted in Fig. 2 that the sodium load given on the sixth day was in all three cases too small to replace the total amount of sodium lost in the urine in excess of the intake. Increased return was found on the seventh day, and on the eighth day it was approximately 100 per cent in all three individuals. R. M. showed a weight increase, on the seventh day, of 800 Gm. Subject W. L. exhibited a weight gain of 300 Gm. and Subject W. C. of

600 Gm. In both of these latter cases, the return of sodium was such that it practically equaled the total amount of sodium the subjects had lost in the urine during the entire experiment in excess of the intake, while R. M. had a positive balance of approximately 135 meq. The following day, a third sodium load of the same magnitude was given the same three individuals, the result of which is seen in the third section of Fig. 2. Only Subject R. M. showed a slight weight increase of 100 grams, while Subject W. L. lost 900 grams and Subject W. C. 1,300 grams.

Serum sodium and potassium in those individuals who received a low-sodium diet as well as a daily intake of ammonium chloride were practically unchanged during the whole experiment. The CO_2 dropped steadily and was, on the last day, 18 and 19 meq. per liter in Subjects W. L. and W. C., respectively, and 24 meq. per liter in Subject R. M.

The effect of an oral sodium load given two individuals who had starved for two and three days, respectively, is finally shown in Fig. 3. Subject W. C. (T in Table I) received on the third day of starvation a sodium load, which, as can be seen, was considerably higher than the total amount of sodium lost in the urine in excess of the intake. The percentage return of the sodium load was only approximately 20 per cent. In J. B. (U in Table I), who received a similar amount of sodium, the return was even less than one-half of that seen in Subject W. C. Both of these subjects lost 1,100 grams.

The students who were on starvation showed practically no change in serum sodium or potassium during the period of the experiment. In none of the individuals who were given a water load (Table I) was any difficulty found in inducing water diuresis. However, it should be emphasized that water loads were only given prior to sodium loads or after signs of sodium retention had ceased.

Finally, one subject, K (Table I), was, after eight days of a low sodium intake, given 15 Gm. of ammonium chloride during one day. The serum sodium prior to this ammonium chloride load was identical with the serum sodium found on the day the experiment started (137 meq. per liter). However, the next day it had dropped to 132 meq. per liter and the following day it dropped to 123 meq. per liter. The minute amount of sodium excreted in the urine on the day of the ammonium chloride load was practically unchanged. while the following day it increased 9 meq. The potassium excretion, however, increased 37 meq. The excretion per minute the day following the ammonium chloride load was for sodium, 26 microequivalents per minute, which was an increase of 23 microequivalents per minute, and for potassium 94 microequivalents per minute, which was an increase of 41 microequivalents per minute.

DISCUSSION AND CONCLUSION

The results obtained in this study seem to confirm the observation by Black, Platt, and Stanbury[4] pointing to an obligatory sodium retention in persons who have been on a low-sodium diet for a certain length of time. The results obtained here, however, also seem to indicate clearly that the percentage return

decreases gradually as time goes on. It is, however, very important to notice that the return is down to approximately 20 per cent after only two days of low sodium intake.

It is of interest also to notice that as little as two to three days of starvation is sufficient to decrease the percentage return of a sodium load to something in the order of 10 to 20 per cent. This again may mean that a patient can very easily run the risk of developing a sodium retention prior to an operation.

It is difficult to explain why the percentage return of a sodium load is higher as the ammonium chloride load is increased. Since, however, the ammonia excretion in the urine, which in these cases is already high, does not rise immediately, and since the chloride excretion gradually increases, one may suggest that sodium and potassium are excreted possibly in order to neutralize the excess acid until the ammonia production is adequate to prevent further loss of base. Although only one student was given an ammonium chloride load on the ninth day of his low sodium intake, this experiment seemed to indicate that when no adaptation has taken place, such a sudden acid load can lead to a rather dramatic drop in serum sodium. The mechanism which brings this about is probably to be found in derangement in the cells, since neither the weight change (decrease) nor the sodium excretion can explain such a drop in serum sodium. The increased excretion of potassium, which was in excess of the increase in sodium excretion, also seems to indicate an exchange of potassium for sodium in the cells. Although such an exchange may also take place when a person on a normal diet is given ammonium chloride, it seems as if adequate amounts of sodium are available to prevent any drop in serum sodium. However, this might not be the case in a person who has been on a low-sodium diet for some period of time.

It is surprising to notice that the weight increase found following a sodium load when the percentage return is low is in no way equal to the amount of sodium retained. For instance, S. D. (Fig. 1, F in Table I) on the ninth day of the low-sodium diet was given 265 meq. of sodium, of which he retained practically everything, yet the weight increase was only 600 grams. Since there was no considerable increase in potassium excretion, it must be concluded that the greater amount of sodium must have been deposited in cells or extracellular solids in such a way that it has been deprived of its osmotic activity.

In conclusion, it can be said that if a person is subjected to a restricted sodium intake, his ability to excrete sodium diminishes rapidly, leaving him after a couple of days in a condition where he may be unable to excrete larger amounts of sodium. Unless one is forced to restrict sodium, as in cases of hypertension or heart conditions, it would seem very undesirable to restrict the sodium intake of a patient preoperatively for any length of time, since this may only aggravate a possible postoperative salt retention. On the contrary, it seems indicated to make certain that a patient preoperatively is getting adequate amounts of sodium (as well as potassium) since this may also prevent any extensive drop in serum sodium induced by the stress itself.

SUMMARY

Sodium deficiency was established in twenty-one male medical students.

The first group of students received a low-sodium diet for a varying number of days.

The second group of students received in addition to a low sodium diet a daily intake of 10 to 20 Gm. of ammonium chloride.

The third group of students was starved for three to four days.

The percentage return of an oral sodium load in all three groups showed a gradual drop depending on the length of time they had been subjected to a low sodium intake. The return was independent of the magnitude of the sodium load as well as of the total amount of sodium lost in the urine prior to the load.

It is emphasized that only two days of starvation is sufficient to lower the return of a sodium load to 25 per cent of the normal. The possible significance of these findings for the surgeon in the preparation of a patient for surgery is discussed.

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THE USE OF ARFONAD IN A MODIFICATION OF THE TECHNIQUE OF CONTROLLED HYPOTENSION

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THE problem of excessive bleeding during surgical interventions is scarcely a new one, and for many years a number of anesthetists and surgeons have found a solution in the use of chloroform anesthesia, high spinal analgesia, or the meticulous posturing of the patient so that the operative site might remain superior. In 1946 Gardner[1] introduced the technique of arteriotomy, whereby blood is withdrawn from the patient preoperatively via the arterial route, and then replaced, in part or in toto, by the same manner at the end of surgery. Such preoperative reduction of the total circulating blood volume by the removal of from 500 to 3,000 c.c. of blood serves to lower the arterial pressure to a systolic level of 50 to 90 mm. Hg, and at the same time produces peripheral vasoconstriction[2]; when combined with the proper positioning of the patient, the net result is a reduction of bleeding at the site of surgery. Arteriotomy thus exactly approximates a state of hemorrhagic shock. A more physiologic approach to the problem was contained in Gillies' classic studies in 1948 on total sympathetic blockade.[3] In this method, the blood pressure is reduced to a level of 70 mm. Hg or below by the use of spinal anesthesia to achieve paralysis of the entire efferent sympathetic outflow, with lesser degrees of sensory and motor blockade. Gillies has admitted that even this technique is without doubt "physiologic trespass,"[4] but has assumed that the arteriolar dilatation so produced guarantees normal oxygenation of the tissue cells, and that the decreased cardiac output inherent in the technique is compensated for by the decreased peripheral resistance and, therefore, the decreased work of the heart. Proper posturing of the patient is again an essential feature for the control of bleeding as it is, indeed, in all methods of "controlled hypotension." The introduction of easily injectable ganglionic-blocking agents in the form of the methonium compounds in 1948[5, 6] led to the widespread usage of hexamethonium and, to a lesser extent, pentamethonium, to lower the blood pressure to systolic levels of 50 to 60 mm. Hg and thus control excessive vascular bleeding in the operative field.[7, 8]

There can be little doubt, from the alarming warnings which have been sounded on the subject,[9-12] that certain fatal and nonfatal complications resulting from the use of the technique have been far more common than was previously supposed. Hampton and Little, polling the anesthetists of Great

We wish to express our gratitude to the Medical Department of Hoffmann-La Roche Inc., Nutley, New Jersey, for the generous supplies of the Arfonad employed in these studies.

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Britain and Ireland, obtained reports of over 21,000 cases of controlled hypotension in anesthesia. The results of this questionnaire form the basis of a separate report,[15] but suffice it to say for the present that there was a total of 653 complications and forty-six associated mortalities among the 21,000 cases reported. Furthermore, it has become very clear that the morbidities and mortalities associated with the use of these techniques have been directly related to the extent of the reduction of the blood pressure from the preoperative normal.

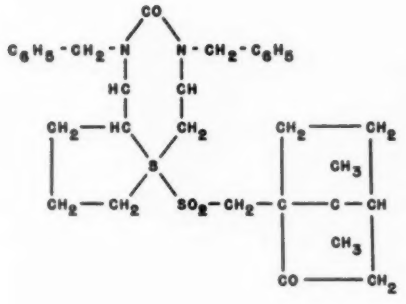
Nevertheless, there also can be no doubt that there are valuable contributions to patient welfare to be derived from controlled hypotension, and that when wisely employed, in the carefully selected case. for an urgent and rational indication, the technique may prove markedly beneficial for the individual patient. Certain definite advantages may accrue from the use of controlled hypotension in anesthesia. Blood loss is tremendously reduced and the patient's own blood thereby conserved, mitigating the need for transfusions with the attendant risks of reaction. Control of vascular ooze provides the surgeon with a clear, almost dry, operating field: this is of utmost concern in an operation such as the Lempert fenestration procedure, in which one drop of blood during the critical maneuver of shaping the fenestra may obscure the entire operative field and prevent a successful result. Finally, the control of hemorrhage, in such operations as the excision of highly vascular intracranial lesions and in certain radical dissections elsewhere in the body, may be essential to surgical success, and operation may be totally unfeasible in the absence of such control.

A MODIFIED CONCEPT OF CONTROLLED HYPOTENSION

Certain important facets of the problem of induced hypotension have been elucidated from the growing body of clinical experience presently extant. First, it has become evident that the level of systolic arterial pressure which must be obtained in order to provide a relatively bloodless operative field varies tremendously with the individual patient, and the exact level to which the blood pressure must be lowered, therefore, cannot be foretold with any degree of accuracy prior to actual operation. It has frequently been advocated that the blood pressure be lowered to, say, 70 mm. Hg in order to prevent vascular ooze in the operative site. Yet in point of fact, in a given patient the blood loss may be controlled only when the arterial pressure has been reduced to 65 mm. Hg; or, conversely, it may be controlled with an arterial pressure as high as 100 mm. Hg. The level that it is necessary to attain can be determined only under conditions of actual operation after suitable posturing of the patient and by direct observation of the bleeding, or lack of it, in the operative field itself. Second, as has just been suggested, this level of optimal pressure is often much higher than the dangerously low systolic pressures of 50 to 60 mm. Hg sought by the earlier workers in this field. Indeed, a significant reduction of the blood loss from the surgical wound is frequently noted at pressures of 90 to 100 mm. Hg, which may represent a lowering of the arterial pressure of only 10 to 20 mm. Hg from the

preoperative reading. This is of some considerable importance, inasmuch as the major complications of deliberately induced hypotension—suppression of kidney function, thromboses of various essential arterial supplies, and reactionary hemorrhage—would not be expected to occur in the absence of profound reductions of arterial pressures. Third, it is essential that the anesthetist be capable of exerting sufficient control over this deliberately produced hypotension to permit its maintenance with some degree of constancy, and yet at the same time to permit the rapid termination of hypotension at the end of surgery or the rapid abandonment of the technique in the face of inadvertent hemorrhage and similar surgical contingencies.

ARFONAD



d-3,4 (1',3'-DIBENZYL-2'-KETO-IMIDAZOLIDO)-1,2-TRIMETHYLENE THIOPHANIUM d-CAMPHOR SULFONATE

Fig. 1.—Arfonad (RO 2-2222).

It is apparent, therefore, that by modifying the current practices in regard to controlled hypotension, it should be possible to obtain the advantages of reduced bleeding during operation without courting the dangers which previously have marred the clinical employment of the technique. There has been lacking a method of sufficient controllability to allow the blood pressure to be lowered gradually during the early moments of the operative procedure, so that the optimal level of the blood pressure at which bleeding in the wound was controlled could be ascertained by direct vision and then maintained by regulating the administration of the hypotensive agent at the necessary rate. This would assure utilization of the maximal level of blood pressure at which excessive bleeding could be controlled.

The introduction of Arfonad, a thiophanium compound with potent ganglionic-blocking activity, has provided the anesthetist with a thoroughly controllable and rapidly reversible method for the induction of hypotension during anesthesia. Chemically, this drug is d-3, 4 (1′3′-dibenzyl-2′-keto-imidazolido)-1, 2-trimethylene thiophanium d-camphor sulphonate (Fig. 1); it is generally referred to by the code number, RO 2-2222, or the trade name,

Arfonad. Randall, Peterson, and Lehmann[13] have reported on the ganglionic blockade and subsequent reduction of arterial pressure produced by this drug in anesthetized dogs and cats, and Sarnoff, Goodale, and Sarnoff[14] have described its use in human beings to produce peripheral vasodilatation and a graded reduction of arterial pressure in the treatment of acute pulmonary edema. It has been shown that Arfonad in single intravenous doses of 0.1 or 0.2 mg. per kilogram of body weight will produce a prompt but evanescent depressor response of the arterial pressure,[14] and it is thus possible to employ the drug by a continuous intravenous infusion technique.

TECHNIQUE

In the present study, Arfonad has been employed in surgical patients to induce a modified form of controlled hypotension for the production of a relatively bloodless surgical field. General anesthesia has been established and endotracheal intubation has been accomplished to assure a patent airway and adequate oxygenation. The endotracheal catheter, in most instances, has then been attached to a nonrebreathing valve to guarantee the removal of all excessive carbon dioxide, the accumulation of which is in itself a potent factor in the causation of excessive bleeding. Following the completion of the anesthetic induction and intubation, the patient has been positioned on the table with meticulous care to place the operative site superiorly, in order to promote venous drainage by gravity from the wound and thus check the amount of bleeding during the surgical procedure. Such posturing of the patient serves the further purpose of pooling the blood in the dilated vessels of the dependent parts of the body, and therefore provides the advantages of bloodless phlebotomy without at the same time incurring the dangerous concomitant of peripheral vasoconstriction. At this juncture the Arfonad infusion in a 0.1 per cent concentration has been started and run at a sufficiently rapid rate to evoke a substantial fall in arterial pressure. The infusion rate necessary to elicit this depressor response, and the extent of the blood pressure fall, effectively establish immediate criteria as to the general susceptibility or resistance of the patient to the drug in the individual case. Furthermore, as the arterial pressure is being lowered, repeated blood pressure readings may be correlated with the degree of vascular ooze visible in the wound in order to ascertain the all-important level of pressure which will provide control of excessive bleeding during operation. The infusion rate may then be regulated to maintain this blood pressure throughout that part of the operation during which it is desirable to control the amount of bleeding.

CLINICAL OBSERVATIONS

The assessment of these facts early in the anesthetic course is well illustrated in Fig. 2: the record of the blood pressure is depicted at the top of the chart, and that of the infusion rate below. It will be seen that three minutes after the Arfonad infusion was begun at a rate of 8 mg. per minute, the systolic pressure had been lowered from 118 mm. Hg to 74 mm. Hg, indicating a substantial susceptibility to the drug. It became clear that

excessive vascular oozing was stemmed during this initial depression of the blood pressure, and the infusion rate was therefore decreased to permit the blood pressure to rise slowly. When the pressure had reattained a systolic level just above 85 mm. Hg, bleeding was once again noted in the operative site; hence, the optimal pressure was established as 85 mm. Hg. The infusion rate was subsequently regulated to maintain this level of blood pressure in order to provide a continuous dry operative field. Nine minutes later, by way of illustrating the prompt resumption of normotension, the Arfonad infusion was discontinued: it is to be noted that preoperative blood pressure had been restored within a ten- to twelve-minute interval.

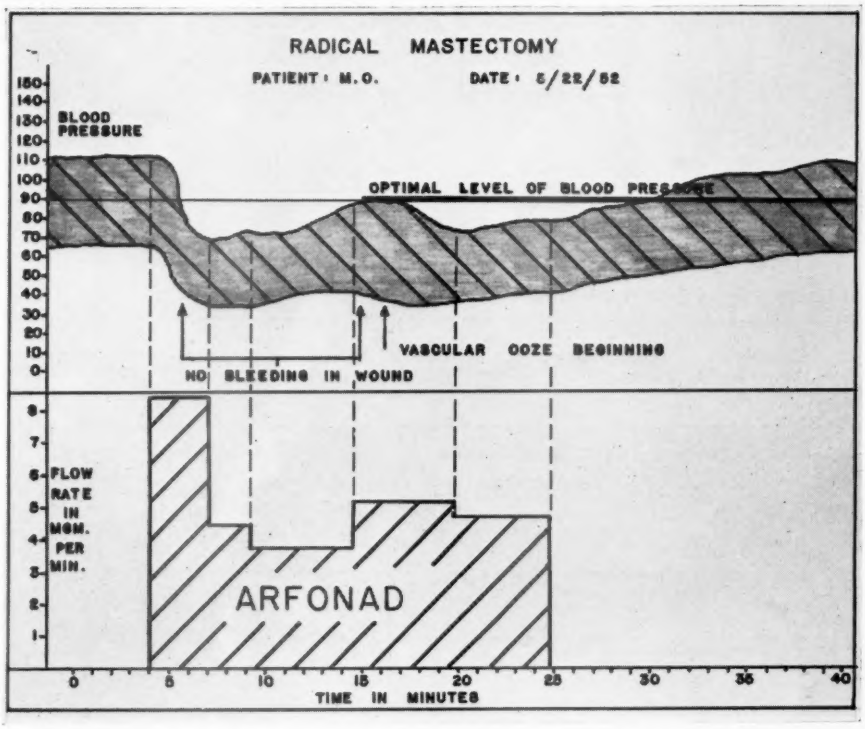


Fig. 2.—Estimation of the optimal blood pressure level at which vascular ooze can be prevented during operation. The blood pressure is lowered slowly by the administration of a dilute intravenous infusion of Arfonad until there is no further bleeding in the operative site, and is then permitted to rise slowly (by decreasing the rate of Arfonad administration) to demonstrate the level of blood pressure at which bleeding recommences.

Such controllability is depicted vividly in Fig. 3. During the initial induction of hypotension, it became evident that bleeding was well controlled at a systolic pressure of 80 mm. Hg. An attempt, therefore, was made to maintain this arterial pressure by regulating the rate of the infusion and thus in effect titrating the patient against the drug: as the infusion rate of the Arfonad solution was increased, the blood pressure fell promptly to lower levels; conversely, as the amount of Arfonad administered was decreased, there was a corresponding and prompt return of the blood pressure toward higher levels. In this manner, it was possible to achieve minute-to-minute control of both the degree and the duration of the hypotension.

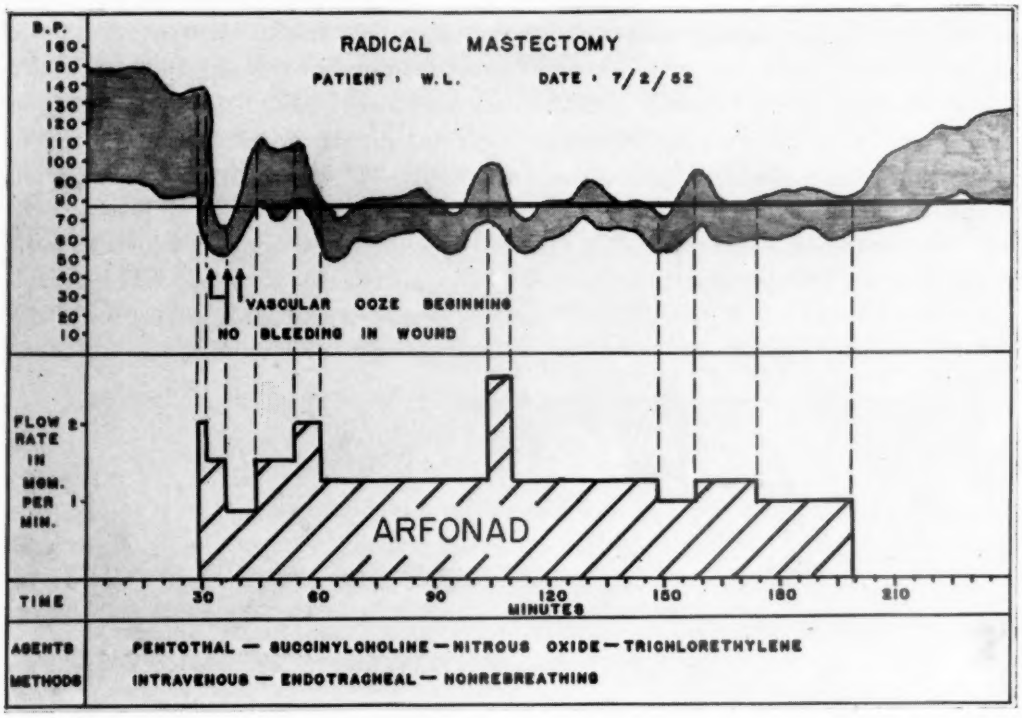


Fig. 3.—The minute-to-minute control of the level of the blood pressure is accomplished by increasing or decreasing the rate of administration of a dilute intravenous infusion of Arfonad.

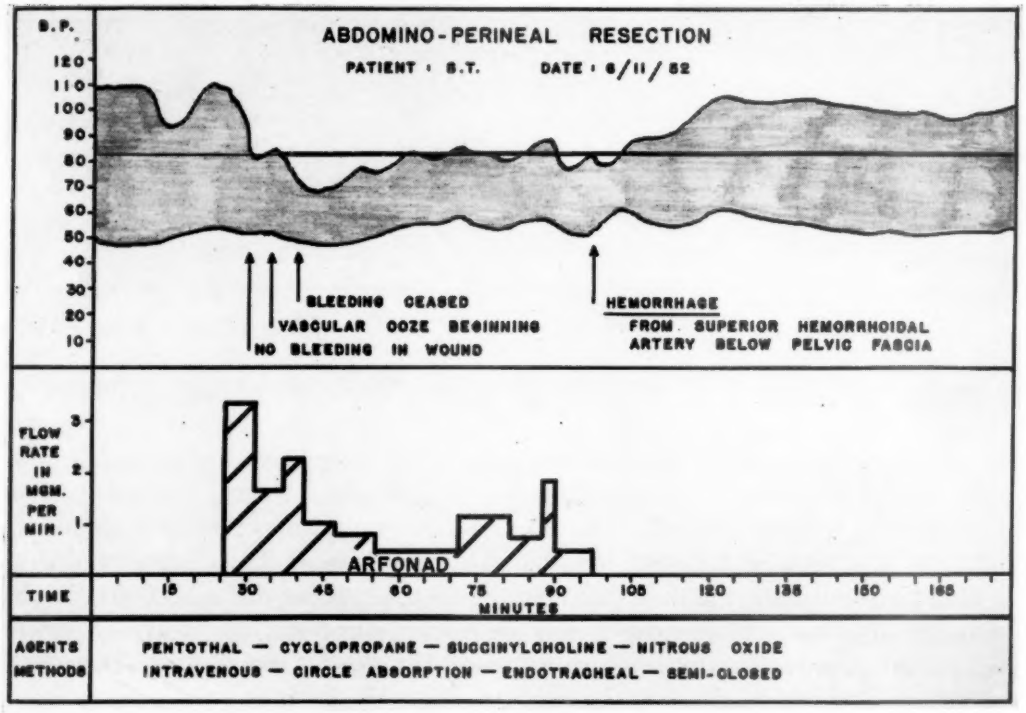


Fig. 4.—The administration of a dilute intravenous infusion of Arfonad may be terminated at the end of surgery or at any other time during operation in the event of hemorrhage or similar surgical exigency, permitting the rapid restoration of normotension.

The termination of this case reveals the advantage of such controllability at the end of surgery, when the need for the hypotensive state has passed and it is desirable to regain normotensive levels. When the infusion was discontinued, there was a prompt rise toward the preoperative blood pressure level. A similar but more dramatic aspect of the advantage of this controllability in facilitating the abandonment of the hypotensive state when necessary is illustrated in Fig. 4. Hypotension was induced, and 95 mm. Hg found to be the optimal level of the blood pressure below which excessive

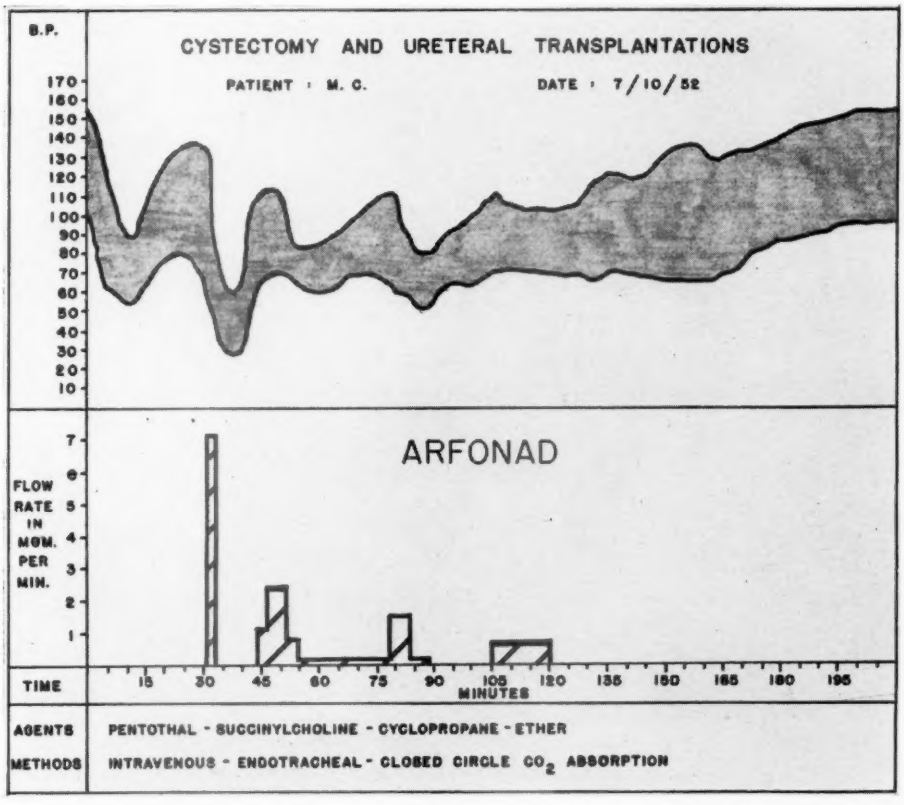


Fig. 5.—Certain patients are extremely susceptible to the intravenous administration of even dilute infusions of Arfonad, to the extent that wide and presumably dangerous swings in blood pressure occur and control becomes difficult.

vascular ooze was well controlled. This level was therefore achieved, and maintained fairly well throughout the first hour of operation. At this point, brisk hemorrhage was encountered from the superior hemorrhoidal artery. The technique of controlled hypotension assumes additional dangers in the presence of hemorrhage, since blood loss is occurring in the presence of generalized arteriolar dilatation and, hence, in the absence of the body's compensatory cardiovascular reflexes. The Arfonad infusion was therefore discontinued, the blood pressure began to rise within five minutes, and the preoperative systolic level of 110 mm. Hg was achieved within twenty minutes.

DISADVANTAGES OF ARFONAD

There are certain disadvantages to the use of Arfonad, some of which are unique to the drug itself, and some of which it shares in common with other methods of autonomic ganglion blockade employed for controlled hypotension. The inherent capacity for catastrophe that is the hallmark of all forms of induced hypotension during surgery has already been mentioned in some detail, and without question applies to the technique herein described as it does to all others. The higher blood pressure ranges utilized in this modified technique, however, and the more absolute controllability possible with Arfonad, reduce these dangers to a very considerable extent.

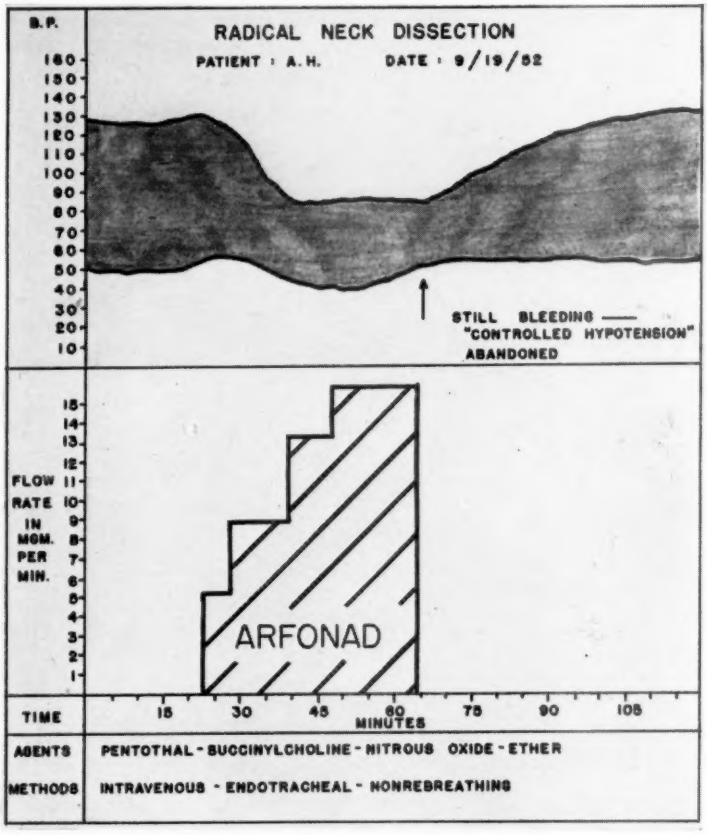


Fig. 6.—Marked resistance to the hypotensive effects of the administration of dilute intravenous infusions of Arfonad has been noted in some patients, and large amounts of the drug may fail to lower the blood pressure sufficiently below normal to effect a diminution of bleeding in the operative site.

Extreme susceptibility to the drug occurred in certain individual patients, just as it does to the methonium compounds, and such extreme susceptibility may exaggerate the technical difficulties to a marked degree. In one instance (Fig. 5), the patient's extreme susceptibility rendered control difficult, and produced wide and presumably dangerous swings in blood pressure.

On the other hand, marked degrees of resistance were encountered also, to the extent that it was occasionally impossible to induce a satisfactory degree

of hypotension and concomitant control over the degree of bleeding in the operative site. Such instances of resistance have been noted frequently to the methonium compounds, particularly in the young and robust, and it remains to determine the exact incidence of such resistance to the employment of Arfonad. During the course of one operation (Fig. 6), a very rapid infusion rate and a total dosage of 500 mg. of Arfonad failed to lower the blood pressure to any marked degree or to achieve any visible reduction of the amount of bleeding in the operative site, and the technique was subsequently abandoned in this instance.

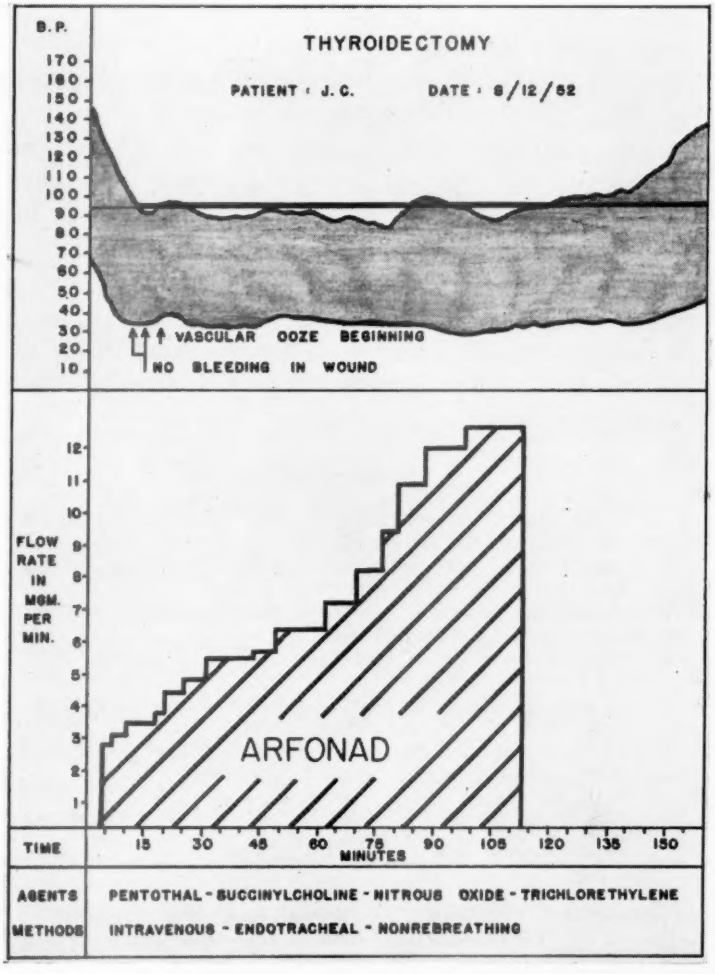


Fig. 7.—Tachyphylaxis has occurred during the intravenous administration of dilute infusions of Arfonad employed to achieve controlled hypotension. In such instances, the optimal level of blood pressure, or below, has been maintained throughout operation, but increasing dosages of the drug have been required to accomplish this.

A final disadvantage to the use of Arfonad was the development of tachyphylaxis on four occasions (Fig. 7). The initial induction of the hypotensive state, and the elucidation of the optimal blood pressure level necessary to control bleeding, were accomplished satisfactorily, but the maintenance of

that level required increasingly more rapid infusion rates and hence higher total dosage of Arfonad. Such tachyphylaxis has not yet progressed to the point at which the blood pressure could not be maintained at the desired level, but this is at least a theoretical possibility.

CONCLUSIONS

Controlled hypotension to provide a bloodless operative field must be assumed to be a gravely dangerous technique, and it has become very clear that the morbidities and mortalities associated with its use are directly related to the extent of the reduction of the blood pressure from the preoperative normal. However, the use of these techniques of induced hypotension in certain specific surgical procedures has permitted successful operative intervention that would not have been possible under other circumstances.

A modified form of controlled hypotension has been evolved whereby a relatively bloodless operative field is produced at higher, and therefore presumably safer, levels of blood pressure than those that have been advocated in the past. The use of Arfonad, which possesses potent but evanescent ganglionic-blocking activity, provides a method of ascertaining the level of the systolic blood pressure at which bleeding is controlled during surgery, and further provides a means of maintaining that level with marked controllability. At the completion of surgery, or in the presence of hemorrhage or similar surgical exigency, this controllability will permit rapid reversal of arteriolar dilatation and the hypotensive state.

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TRANSPLANTATION OF THE SAPHENOUS VEIN FOR POSTPHLEBITIC STASIS

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IN RECENT years, vigorous surgical attacks have been made on various parts of the venous system of the lower extremity in order to improve the lot of individuals suffering from postphlebitic stasis. Superficial femoral vein interruption,[1] popliteal vein interruption,[2] interruption or ligation of communicating veins,[3] or of elements of the saphenous system, alone or in combination, have been recommended with varying degrees of reported success. Femoral vein interruption, although still advocated by some, was the subject of a lukewarm supplementary report by its original supporters,[4] and the initial reports on popliteal vein interruption have not been enthusiastic. Favorable results of such surgery have been reported by Linton,[5] who, using a combination of attacks in increasingly radical operations, has found a large percentage of ulcers to have healed. A significant part of his recommended therapy, however, is the provision of adequate elastic support following surgery.

The concept of removal or interruption of damaged veins arises from the commonly observed excellent results afforded by obliteration of the saphenous veins of patients with varicosities of that system but with intact deep veins. It is entirely possible, however, that deep veins with incompetent valves may not permit such unimpeded backflows as occur in valveless superficial skin veins. Furthermore, it has been suggested that, despite a marked degree of insufficiency during diastole of the "venous heart of the lower extremity" (muscular relaxation of the calf), the integrity of continuity of a principal deep vein may still be important to the lower extremity, since no other main channel may exist for outflow during the systolic calf contraction.

Venous pressures in the skin veins of the calf do not decrease more markedly in postphlebitic syndrome after superficial femoral vein interruption than before it,[6, 7] and in certain instances the postinterruption pressures have been found to have altered in an unfavorable direction.

In light of the unpredictable results following such surgical procedures as have been listed, it seems logical to search for a means of attack which would provide new or improved channels of venous return rather than to concentrate entirely on destruction of possibly useless, but probably harmless, ones already existing. In March, 1949, therefore, a transplantation of the saphenous vein was performed to the muscular compartment of the thigh of

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a 48-year-old man apparently suffering from postphlebitic syndrome as a result of phlebitis following a back injury six months before. The saphenous vein was completely freed throughout the thigh, all branches being divided, leaving it, however, attached to the femoral vein above and to its connections to the calf below. It was then buried over as great an extent as possible beneath the deep aponeurosis of the medial aspect of the thigh. Following the procedure the walking venous pressure showed improved function by a 14 cm. further fall during walking over the preoperative value, and phlebography showed the vein to have remained patent. Despite the fact that in this case a later phlebogram disconcertingly revealed a relatively normal original femoral vein, it is believed that the patient's steady clinical improvement since that time was at least partially due to his ability to use more effectively the saphenous transplant in the thigh. It is assumed that the area of greatest postphlebitic damage in this patient was in the calf veins. Since that time thirteen additional similar operations have been performed.

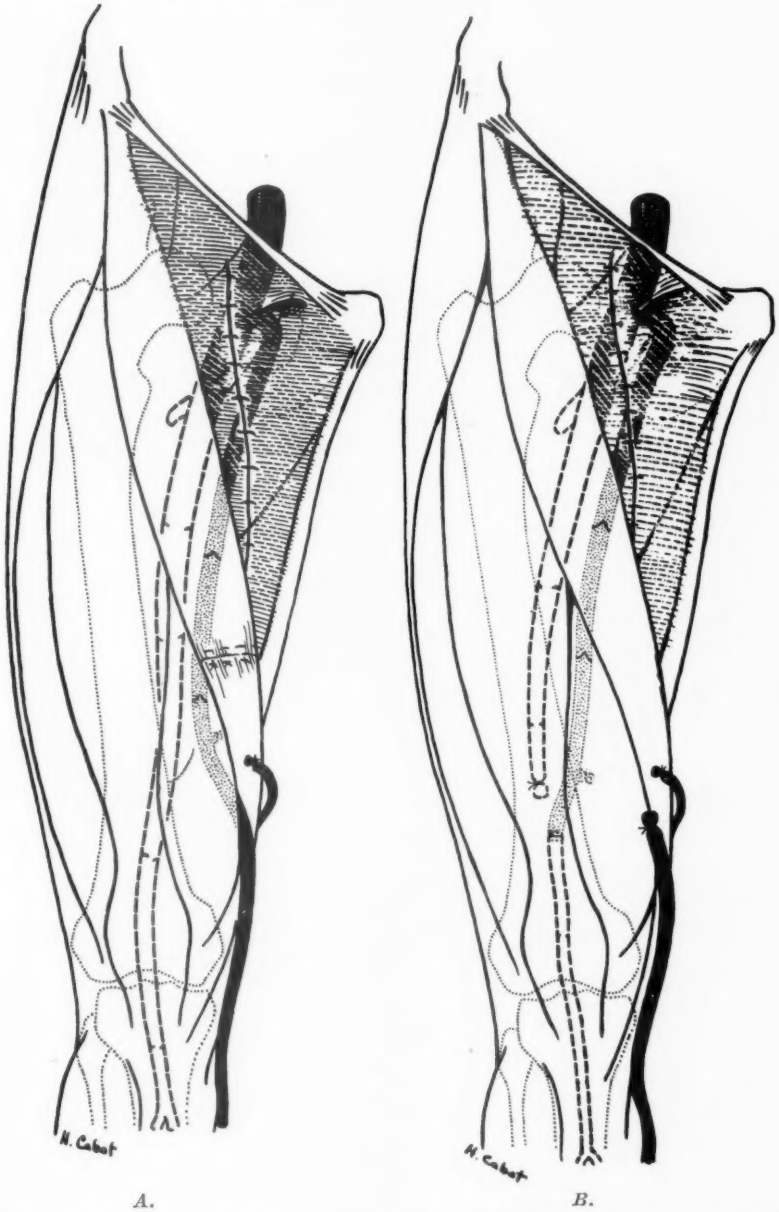
TECHNIQUE

An incision is made on the medial face of the thigh from groin to knee over the course of the previously identified internal saphenous vein. The vein is carefully freed from all its attachments other than those to the femoral vein above and to the calf veins below. All branches are ligated flush with the main vein.

In the cases reported, one of two general forms of operation was then undertaken. In the first form, the saphenous vein, so freed, was transplanted to the muscular compartment of the thigh without surgical manipulation (Fig. 1, *A*). In the second form, the same transplantation was accomplished but, in addition, the vein was divided at the knee and an anastomosis performed between the proximal segment of the saphenous and the distal segment of the divided popliteal vein (Fig. 1, *B*).

Incision of the aponeurosis between the adductor muscles of the thigh and the extensors of the knee exposes the femoral vessels and permits the saphenous vein to be transplanted to lie alongside them. The aponeurosis is closed over it taking care to avoid kinks in the transplant. In the last three of the seven transplants which were performed without an anastomosis, the sartorius muscle was divided and resutured over the transplanted vein, thereby allowing the latter to be buried over a longer distance in the thigh than in the earlier cases. In the case of the transplants with anastomosis, the freed long saphenous vein was divided at the level of the knee and the distal cut end of the proximal segment drawn through under the sartorius muscle into the popliteal space, where, having dissected out and divided the popliteal vein, an anastomosis between the distal cut end of the proximal segment of the saphenous vein and the proximal cut end of the distal segment of the popliteal vein was carried out. In five of the seven cases in which this was done, the remaining saphenous vein in the calf was excised either by stripping or by direct excision. In the other two cases, the proximal cut end of the distal segment was simply ligated. The distal cut end of the proximal segment of the popliteal

vein was ligated in all cases. The transplanted saphenous vein was in most of the cases brought around, or medial to, the tendon of the adductor magnus rather than through the muscular canal, where there seemed to be little room for it unless created by a traumatic dissection. The popliteal vein in all cases



A. *B.*

Fig. 1.—Diagram of disposition of saphenous vein following transplant. *A*, Without saphenopopliteal anastomosis. *B*, With saphenopopliteal anastomosis.

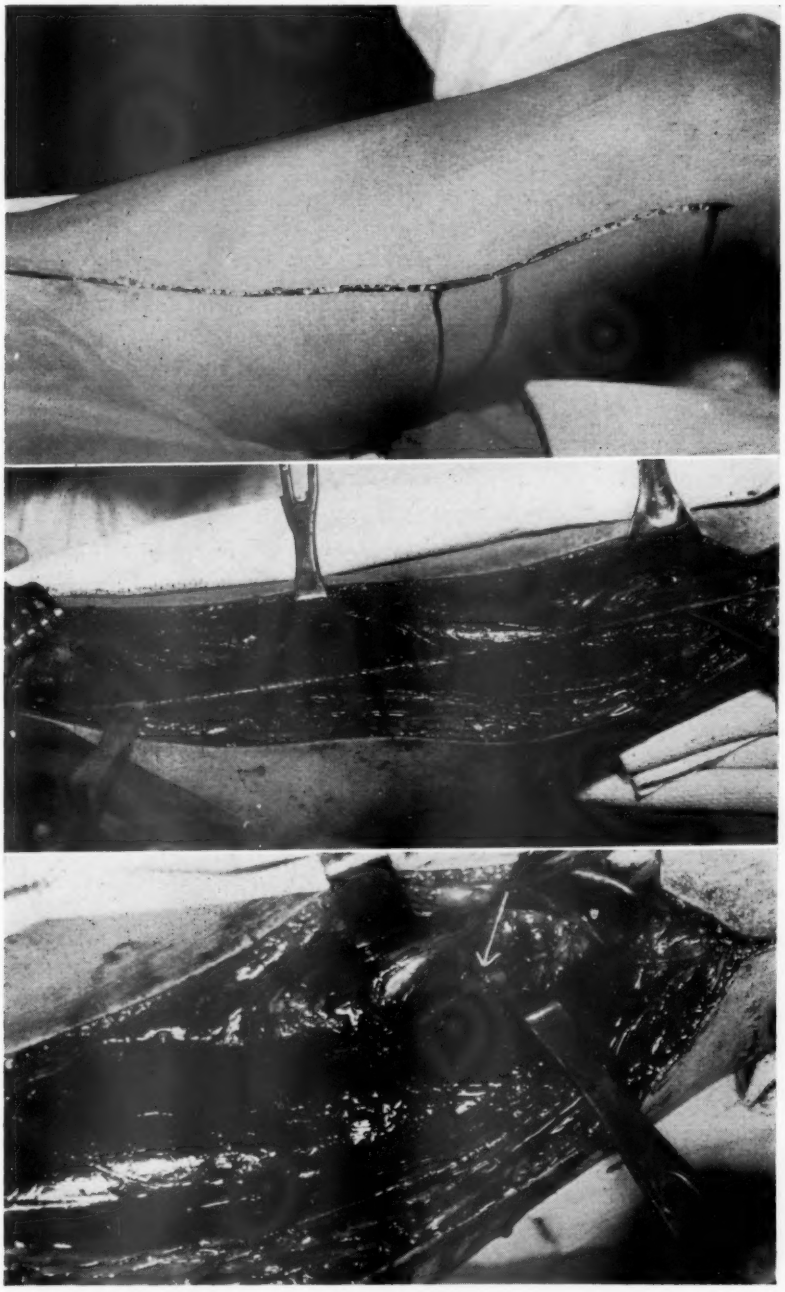
showed evidence of recanalization, a feature which made the anastomosis difficult and may be a reason for failure in some. In all instances but one, the saphenous vein was anastomosed to the whole circumference of the popliteal vein, which in its recanalized state presented two or more lumens. In one

TABLE I. CASE SUMMARIES OF SAPHENOUS VEIN TRANSPLANTS: OPERATIVE DETAILS

PATIENT	SARTORIUS MUSCLE DIVIDED	SAPHENO-POPLITEAL ANASTO-MOSIS	FEMORAL VEIN DIVIDED	POPLITEAL VEIN DIVIDED	SAPHENOUS VEIN EXCISED IN CALF	REGIONAL HEPARIN-IZATION	LENGTH OF FOLLOW-UP (MO.)	CLINICAL RESULT	WALKING VENOUS PRESSURE RESULT	VENOGRAPHIC RESULT
J. M. D.	0	0	0	0	0	0	48	Improved	Improved	Open
T. V.	0	0	+	0	0	0	48	Unimproved*	Worse	Open
E. F. W.	0	0	+	0	0	0	42	Improved	Not carried out	Open
E. F. G.	0	0	0	0	0	0	6	Improved†	Improved	Open
J. J. B.	+	0	+	0	0	0	6	Improved‡	Improved	Open
E. E. M.	+	0	+	0	0	0	6	Improved§	Improved	Open
J. E. McA.	+	0	0	0	0	0	30	Unimproved‖	Improved	Open
C. A. M.	0	+	0	+	0	+	27	Improved	Same	Open
A. McG.	0	+	0	+	+	+	24	Improved	Improved	Questionable
D. M.	0	+	+	+	+	+	23	Unimproved¶	Improved	Not visualized
A. R.	0	+	0	+	0	+	22	Improved	Improved	Not visualized
A. A.	0	+	0	+	+	+	17	Improved	Same	Questionable
H. R. C.	0	+	+	+	+	+	15	Unimproved¶	Improved	Not visualized
F. J. I.	0	+	+	+	+	+	12	Improved	Worse	Not visualized

*Recently (April 23, 1953) had further surgery—saphenopopliteal anastomosis and excision of calf varices.
†Died of "cerebral hemorrhage" ten months postoperatively.
‡Further (three-year) follow-up report (patient not seen) is that he is working full time in restaurant.
§Died twenty-two months postoperatively—coronary heart disease with myocardial infarction.
‖Recently (Feb. 24, 1953) had further surgery—saphenopopliteal anastomosis and excision of calf varices.
¶Femoral vein divided at previous operation.

case, a thick partition separated a large lumen from a very small one, and here the saphenous vein was sutured to the large lumen alone, the smaller lumen being obliterated by sutures.



A.

B.

C.

Fig. 2.—Photographs of operative procedure with saphenopopliteal anastomosis. *A*, Incision. *B*, Prepared saphenous vein. *C*, Anastomosis of saphenous and popliteal veins.

Regional heparinization was used in all the seven patients in whom an anastomosis was performed. One hundred milligrams of heparin in a twenty-four hour period (approximately one-third the amount necessary to maintain

systemic "heparinization") was dripped into a polyethelene catheter tied into the short saphenous vein near the popliteal vein. This twenty-four hour ration was in most cases given in 1,500 c.c. of fluid.

RESULTS

A summary of the results is provided in Table I.

Evaluation of Clinical Results.—Ten of the fourteen patients demonstrated improvement at the time of follow-up examination in that those who had had ulcers showed them to be still healed or, having recurred, had done so with less severity and often a longer period of freedom than had been the case preoperatively. In those who had not suffered from ulcers, the criteria for improvement were diminution in dermatitis or edema. No patient was cured.

The good clinical results were distributed evenly between the two types of operation. The results in the group with anastomosis are open to some criticism as an evaluation of the transplanting procedure itself, since procedures other than transplantation were carried out at the same time. Popliteal vein division was, of course, performed in all seven, and in the majority the saphenous vein in the calf was simultaneously excised because of the existence of large varices. Furthermore, one might state that in the cases in which the transplanted vein was not proved to be patent an effect similar to obliteration of that vein was produced, a matter which would appeal to the supporters of obliteration operations.

TABLE II. WALKING VENOUS PRESSURE BEFORE AND AFTER SAPHENOUS TRANSPLANTATION

PATIENT	PREOPERATIVE VALUE* (CM.)	POSTOPERATIVE VALUE (CM.)	CHANGE (CM.)	RESULT
J. M. D.	+2	-20	-22	Improved
T. V.	-3	+5	+8	Worse
E. F. W.	-7	Not carried out		
E. F. G.	-10	-25	-15	Improved
J. J. B.	-14	-28	-14	Improved
E. E. M.	-1	-14	-13	Improved
J. E. McA.	-16	-19	-3	Same
C. A. M.	-1	0	+1	Same
A. McG.	+3	0	-3	Same
D. M.	-5	-23	-18	Improved
A. R.	-3	-19	-16	Improved
A. A.	-8	-8	0	Same
H. R. C.	-3	-8	-5	Improved
F. J. I.	-7	0	+7	Worse

*The figures represent the change in pressure in the saphenous vein of the calf during walking from that existent when the individual was standing quietly erect.

Evaluation of Walking Venous Pressures.—Table II lists the values for walking venous pressures observed pre- and postoperatively. Pressures were recorded in the internal saphenous venous system as described by one of us (R. W.) and co-workers in 1949.[6] In seven of the cases, the changes in walking venous pressure toward improvement or unimprovement conformed with the clinical results observed. In seven cases in all, the changes were in the direction of improvement. It is to be noted that in Table II no case was classified

A.

B.

Fig. 3. (Case C. A. M.).—*A*, Ulceration of medial aspect of left ankle before operation. *B*, Healed incision and area of healed ulcer twenty-seven months after saphenous transplant with saphenopopliteal anastomosis.

as improved or unimproved unless the change over the preoperative value was more than 5 cm. It is our opinion that the walking venous pressure test, being an objective means of evaluation, is more indicative of the function of the venous return of the extremity than is the clinical result, which can be affected by the activity of the patient and the care with which he uses elastic support.

A. *B.*

Fig. 4 (Case E. F. W.).—Transplantation of saphenous vein without anastomosis. *A*, Preoperative phlebogram. *B*, Postoperative phlebogram illustrating transplanted saphenous vein.

Evaluation of Phlebograms.—Pre- and postoperative phlebograms were made in all cases. In the seven patients who had saphenous transplants without anastomosis, the transplants were readily recognized as being patent postoperatively in all (Fig. 4). In the seven patients who had transplants with anastomosis, the phlebograms were equivocal. In patients with venous stasis, many skin veins exist in the popliteal area, any one of which may be mistaken for the transplanted vein. In some of the patients with anastomosis, an apparently patent saphenous transplant was seen, but the area of the anastomosis was narrow or invisible. In only one of the seven can it be said that a definitely patent anastomosis was seen.

Complications.—In two patients, there were major complications. In one, the medial skin flap became necrotic over a strip 10 cm. long and 3 cm. wide. Eventual healing without disability occurred. In another patient, the symptoms and signs of venous stasis became acutely worse shortly after discharge from the hospital twenty-one days after operation. Dilated skin veins and massive edema appeared. It is probable that a recurrence of deep phlebitis with extension into collaterals has supervened. This patient is one of our poor late results on all counts.

COMMENT

The principle of the operation described is not a new one. Katzenstein[8] in 1911 stated that he had for two years been performing an operation which transplants the largest veins into the deep lying musculature, and stated that an Italian surgeon had been performing a similar one. Apparently Katzenstein contrived a channel in the sartorius muscle by folding its edges together. He stated that the symptomatic results were very good even when the calf veins did not entirely disappear. It is apparent from the article that he did not differentiate between disease of the saphenous system alone and that which resulted from disease of the deep system. Numbers of cases are not given, and later reports have not been discovered.

It is appropriate to discuss the possible directions in which future progress in this field might be made. Since the disease in many patients lies more in the calf than in the thigh, thought should be directed toward applying the principle of this type of operation at points more distal than has been done in the cases described. Since an important concept behind the operation as performed is that in postphlebitic syndrome the majority of saphenous trunks in the thigh maintain their valves, further attempts should be made to ascertain whether this is so. Heretofore, fear of thrombosis has deterred manipulation or injection of the transplant to an extent which would provide this information. If such is not the case, free transplantation of healthy valved veins from elsewhere in the body must be considered. The problem of maintaining rapid blood flow through the anastomosis is a real one. More rapid infusion with dilute heparin solution than has been reported should, we believe, be used.

SUMMARY

1. A report is made of fourteen patients with postphlebitic syndrome who were treated by transplantation of the internal saphenous vein of the thigh

from the subcutaneous to the subaponeurotic area. In one-half of the patients, concomitant anastomosis of the lower end of the saphenous vein to the popliteal vein was carried out, and in some of these, also, the saphenous vein was excised from the calf.

2. Ten of the fourteen patients were clinically improved.

3. Objective evaluation of the patients has been difficult. All the transplants without saphenopopliteal anastomosis were shown to be patent at phlebographic follow-up study. This was true of only one of seven patients having transplantation with anastomosis. A fair correlation between these facts and clinical and ambulatory venous pressure improvement was evident.

4. Suggestions as to causes for failure and comments on possible lines of future progress are made.

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A TECHNIQUE FOR FACILITATING THE STRIPPING OF LARGE-BORE VARICOSE VEINS USING CONVENTIONAL STRIPPERS

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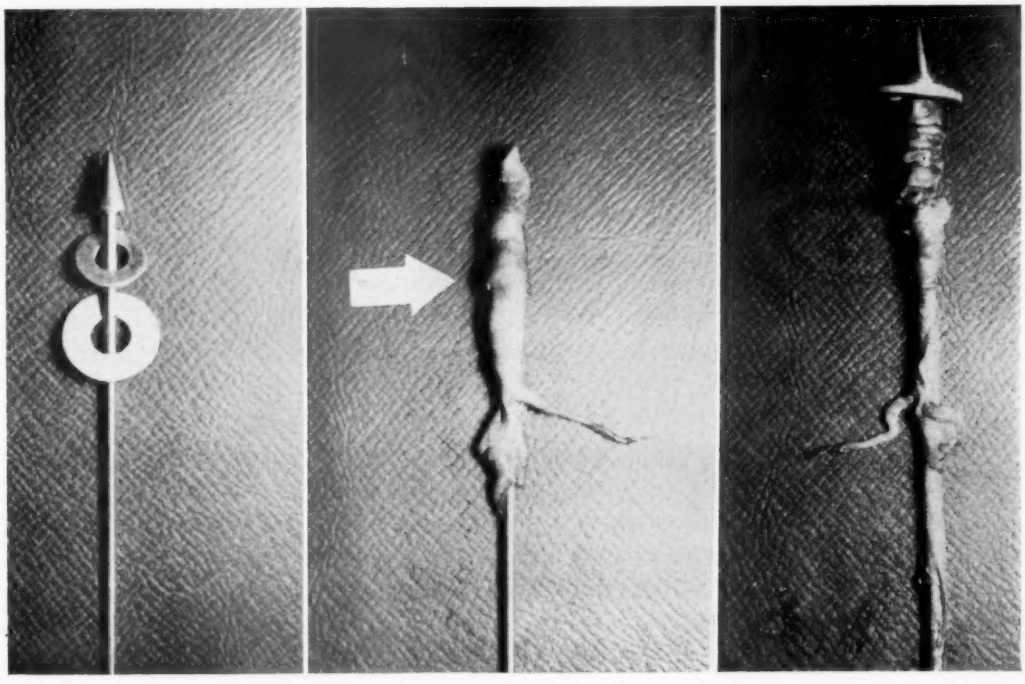
THE technique of high ligation of the greater saphenous vein and its tributaries with stripping of the vein from groin to ankle is widely accepted at present as the preferred treatment for most patients with varicose veins of the lower extremities. When carried out in the usual case without difficulty, this technique permits the avulsion of the entire length of vein between upper and lower incisions. Quite often, because of undue tortuosity or catching of the stripping instrument in a valve or at a bifurcation, the employment of auxiliary incisions and successive segmental stripping maneuvers becomes necessary.



A. *B.* *C.*

Fig. 1.—

A, Babcock type of intraluminal vein stripper with metal "washers" in place near larger end of the instrument.

B, Inversion of large-bore vein by Babcock stripper (without metal washer), as instrument is pulled into lumen. Further traction (in vivo) would lead to tearing of vein. *Arrow* points to head of instrument inside vein.

C, Pleating of large-bore vein on stripping instrument with inversion of vein prevented by metal washer.

An important difficulty arises in the case of "large-bore" veins when the stripping instrument, instead of pleating the vein on itself as traction is begun, inverts the vein as the head of the instrument slips through the large,

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distensible lumen (see Fig. 1, *B*). This may occur even though the vein is securely ligated to the instrument. Further traction, in such an eventuality, results in tearing of the vein wall, usually at the point of entrance of a tributary vein or where the wall of the vein is thin, and the stripper is drawn through with only one or two inches of vein attached. As seen in Fig. 1, *C*, this is not likely to occur if pleating of the vein along the stripper takes place, since the weakest point now becomes the tributary branch rather than the varicosed saphenous vein.

There are some stripping instruments made with detachable metal tips so that large-bore veins may be properly stripped by affixing a tip of suitable diameter. However, a conventional vein stripper of the Babcock or Linton type may also be modified by the use of ordinary metal "washers" of various sizes, which may be placed on the stripper before its introduction into the vein (see Fig. 1, *A*). Pleating of the vein is thereby assured and the likelihood of inversion obviated. These washers should preferably be of stainless or chromium-plated steel.

In a number of clinical cases with large varices this technique has proved satisfactory.

SUMMARY

A technique is described for facilitating the stripping of large-bore varicose veins, using conventional strippers.

A SUITABLE TECHNIQUE FOR TOTAL ARTERIALIZATION OF THE DOG LIVER

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IN ORDER to study the effects of both increased blood flow and oxygen tension on hepatic regeneration in the dog, it became necessary to evolve a technique for total arterialization that accomplished these functions satisfactorily and was relatively easy and safe to perform.

Several investigators have reported on their efforts to anastomose the hepatic artery to the portal vein, or one of its major tributaries.[1-3] For the most part, they have been unsuccessful. The most recent report of this maneuver, by Schilling,[4] indicates good results in four animals. Here, portal blood is not diverted away from the liver, so that some dilution takes place with that from the hepatic artery. To date, no quantitative data have been presented to indicate by what magnitude hepatic blood flow is actually increased by this method.

Shortly before the publication of a technique for arterialization by Cohn,[5] we began to use the method to be described here to eliminate all venous blood flow through the liver and to supply it with an increased flow of arterial blood.

TECHNIQUE

The day preceding arterialization, under Nembutal anesthesia, the left jugular vein is removed. As much length as possible is obtained (average 6 cm.). It is then stored in the icebox, in a sterile stoppered flask containing Ringer's solution and penicillin, until used.

Mongrel dogs of both sexes, ten to fifteen kilograms in weight, are anesthetized with either drop ether or intravenous Nembutal (depending on the capabilities of the anesthetist). A small sandbag, or its equivalent, is placed under the back of the animal just below the costal margin to thrust the vena cava and portal vein forward. For the best exposure, a large incision beginning at the midline below the xiphoid is extended in a gentle curve laterally to just beyond the right nipple line, and then caudad. To simplify the procedure, an adequate incision is essential. Wide retraction of the incision with a self-retaining retractor is helpful. The small and large intestines are wrapped in the duodenal mesentery and packed off to the left, thus exposing the portal vein and inferior vena cava.

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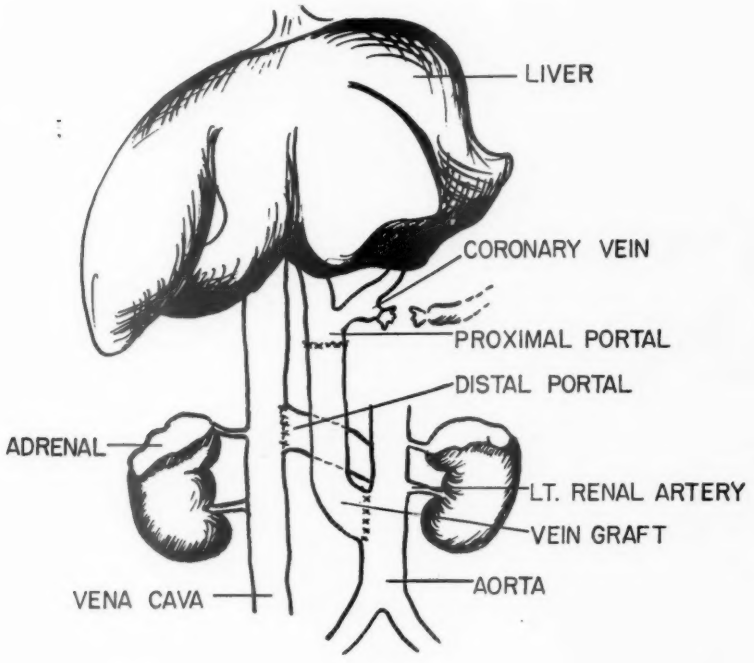
The portal vein is completely mobilized from the point of its bifurcation at the liver hilus to its origin at the junction of the superior mesenteric and splenic veins. Almost invariably, only two branches need be ligated, the coronary vein which usually enters the portal close to the hilus just below the bifurcation, and a very small fine tributary entering at about the middle of the course of the portal vein. If this is not tied, annoying bleeding is likely to occur. With proper exposure and retraction, the entire portal vein may be mobilized from its bed in a few minutes.

Temporarily, the portal vein is gently retracted to the left. This is done so as not to obstruct the flow through it or the hepatic artery. The hepatorenal ligament is severed, and the right liver lobes are retracted laterally, usually by an assistant's fingers over a sponge. The liver must not be traumatized during this procedure. The vena cava, now in full view, is clamped between the right renal vein and the liver by a Satinsky or any other partial occlusion clamp. After an opening is made in the occluded portion of the vena cava and sutures are placed at either end, the mobilized portal vein is once more exposed and severed between bulldog clamps, leaving adequate cuffs of vein beyond both the proximal and distal clamps. The distal lumen of the portal is then approximated to the orifice in the vena cava and, using 00000 silk on No. 9 atraumatic needles, an anastomosis is effected with over-and-over continuous stitches. With the release of the caval and bulldog clamps, there is immediate restoration of the normal pink color of the intestines, which become congested during portal occlusion. The portal vein is usually not occluded for more than ten to twelve minutes. In our hands, the success of this entire operation, just as in total hepatectomy, depends on the facility with which the portacaval shunt can be carried out. Bleeding from the anastomotic line is readily controlled in a few minutes by gentle packing of sponge gauze against it.

Next, the previously removed vein graft is anastomosed to the proximal end of the portal vein. This is accomplished by dividing into four parts the lumen of the portal and anastomosing the vein to it, interrupting the continuous sutures in four places so that little or no purse stringing occurs. As the external jugular vein of the dog has two and sometimes three sets of valves, it is important that the vein be placed so that valvular action will permit the flow of blood toward the liver. Finally, the aorta just below the renal arteries is easily mobilized by spreading the parietal peritoneum and preaortic connective tissue. It is dissected free for a length of approximately 3 cm. Usually, one pair of lumbar arteries must be ligated and severed. In close proximity to the aorta at this level are large lymphatic vessels which cannot be easily separated from the aorta without being torn. They are clamped and ligated wherever possible, although no great effort is made to do this, as invariably the flow of lymph will cease by the end of the operation. The aorta is then clamped by two rubber-shod hemostats which have had their serrated surfaces filed smooth. A slit is made between the occluding clamps about one-half inch long, and small elliptical snips are removed from

each side to make the opening ovoid. The distal end of the graft is now anastomosed to the aorta, using similar suture material and the same technique as for the portacaval shunt.

Upon completion of the graft-aorta anastomosis, first the bulldog clamp is removed from the proximal portal vein, then the distal aorta clamp and, last, the proximal clamp are released. At once, the vein graft is seen to become distended with blood and, on occasion, to pulsate. A thrill is always obtained by gently compressing the graft between the thumb and index finger. Leaks in suture lines are repaired, and the operation is terminated. The completed procedure is diagrammatically demonstrated in Fig. 1.



ARTERIALIZATION OF THE DOG LIVER

Fig. 1.—Diagrammatic presentation of technique for arterialization of dog liver.

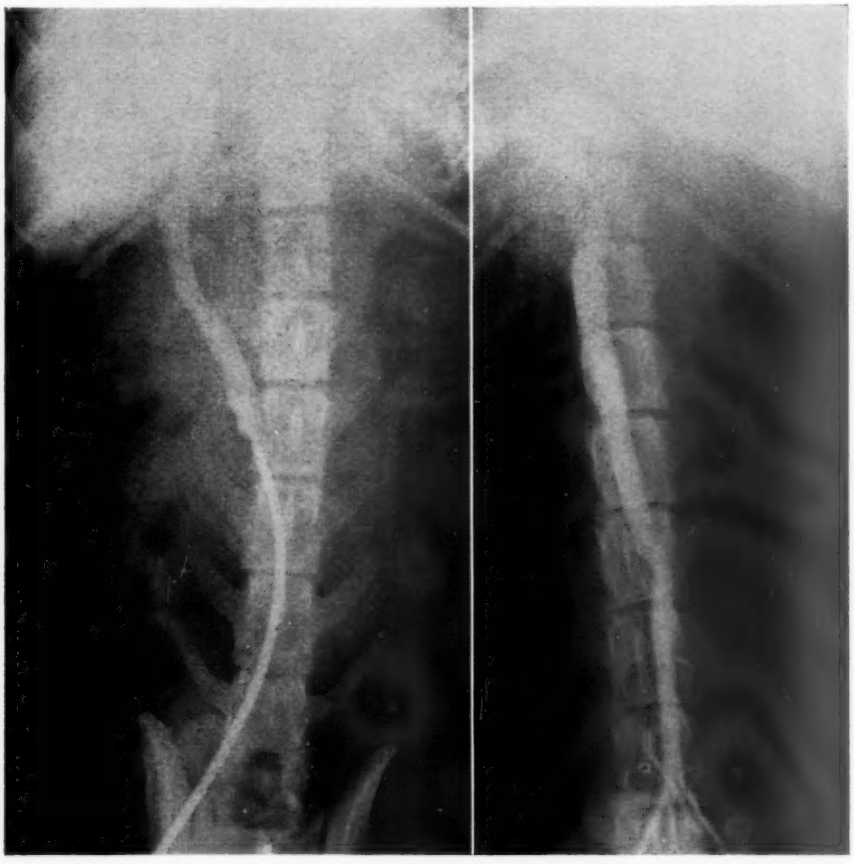
The liver immediately becomes orange-red in color, and the edges are slightly rounded with the interlobar fissures becoming more prominent. In no instance in our experience has the liver pulsated.

The entire operative procedure takes from one to one and one-half hours, during which time the animal receives approximately 150 to 200 ml. of saline solution and whole blood replacement for any blood loss. This is usually small, and under 100 ml.

DISCUSSION

By the method presented, the total blood supply to the liver is arterial. Furthermore, an increase in total hepatic blood flow occurs.

The method described by Cohn has, in our hands, resulted in several technical difficulties. Here, the vein graft is passed through the diaphragm and anastomosed to the thoracic aorta. Frequently we encountered kinking of the graft with blocking of free flow of blood through it, in spite of satisfactory anastomoses. Furthermore, as the graft went through the diaphragm it would occasionally be pinched off by respirations. Third, in our experience at least, partial occlusion clamps applied to the dog's aorta are unsatisfactory, as they do not permit an adequate cuff for a clean anastomosis. Thus, it is necessary to clamp the aorta completely while anastomosing the graft to it. Total occlusion of the thoracic aorta frequently results in hind leg paralysis



A. *B.*

Fig. 2.—Angiograms demonstrating patent channel after (*A*) four weeks, (*B*) six weeks, (*C*) six weeks, and (*D*) eight weeks. (Note constriction at junction of graft and portal.)

of the dog, as has been amply demonstrated by Blalock[6] and Gross[7] in their early studies on the surgical treatment of coarctation. This has been our experience when the aorta at this level is totally clamped for as short a time as fifteen minutes. Clamping the aorta between the renal arteries and trifurcation in twenty-three dogs for from ten to twenty-eight minutes, with an average time of eighteen minutes, has resulted in paralysis or weakness of the

hind legs in no instance. The average time of occlusion in the most recent fifteen animals is twelve minutes, but in earlier operations, occlusion was effected for longer periods of time.

In twenty-seven attempts to carry out this technique, success was obtained in twenty-three animals. In the last fifteen operations, there was only one death, which was due to kinking of the portal vein following portacaval shunt, resulting in portal block and death within eight hours. The cause of death in the other three was hemorrhage in two in the immediate postoperative period and undetermined in one, when the animal was found dead in its cage seventy-two hours after operation.

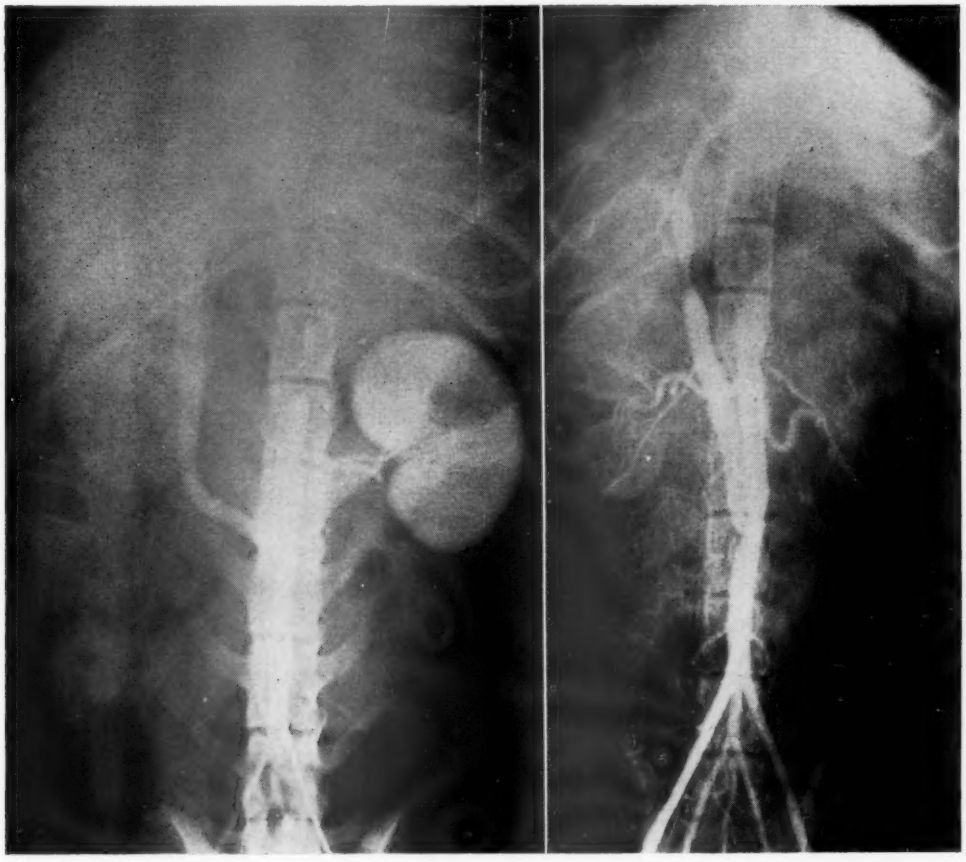


Fig. 2, *C* and *D*. *(For legend see opposite page.)*

In no animals observed did there occur postoperatively dilatation of the vein graft, but some ballooning of the proximal portal vein was common, occurring almost immediately after operation.

Angiograms were made. Fig. 2 shows representative examples demonstrating the new channel. One of the earlier operations, Fig. 2, *D* shows a marked stricture at the graft portal junction after eight weeks, but still permitting blood to travel through it.

Fig. 3 shows a specimen from an animal autopsied two months post-arterialization.

Blood flow studies, oxygen utilization of the liver under these conditions, effects on regeneration, and other studies will be presented subsequently.

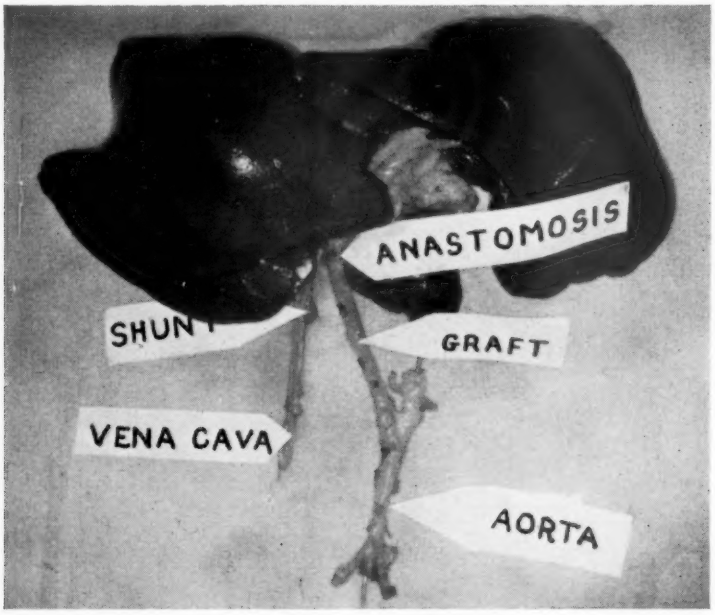


Fig. 3.—Autopsy specimen two months postarterialization.

CONCLUSION

A method for complete arterialization of the dog liver, with low mortality (under 10 per cent in the last fifteen animals) and no morbidity, is presented. This technique is serving as a useful tool for studying the effects of increased blood flow and oxygen environment on the liver.

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RENAL COMPLICATIONS FROM AORTOGRAPHY*

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DAMAGE to the kidney from translumbar aortography has been observed in seven patients in the last two years. During this time, approximately 250 aortograms have been taken on the combined University of California services, of which over 90 per cent were made by the surgeons of the vascular disease group. The seven known instances of renal complications thus represent an incidence of almost 3 per cent.

Since Dos Santos'[1] original warnings about the possibility of renal damage, no serious complications have been reported from aortography, particularly as far as the kidneys are concerned. Minor complications were described in 1952 by Larsson and Palmlöv,[2] who stated that renal injury might result if all or most of the solution were injected into one renal artery. They discussed a patient in whom this was done with subsequent temporary fall in renal function. Another patient received an excess of dye in the renal arteries without apparent damage. Melick and Vitt[3] reported injecting the media directly into the left renal artery, which caused temporary pain and microscopic hematuria, but renal function was subsequently normal.

This report is an analysis of cases of serious renal injury and of data from animal experiments, in an attempt to find the factors causing damage following aortography and the methods for avoiding injury.

From a theoretical standpoint and from an analysis of the clinical data, the cases fall into two main groups: I, direct trauma, and II, indirect damage. These have been further divided:

I. Direct trauma (unilateral or bilateral)
   - A. Misdirected needle
   - B. Excessive pressure/volume
   - C. Chemical irritation from radiopaque media

II. Indirect damage (bilateral, ? reflex)
   - A. Drug toxicity and/or idiosyncrasy (Diodrast, iodine)
   - B. Neurogenic factors?

Clinical Series.—

I. On analysis, three of our seven patients fit into Group I. The damage in all of these was unilateral. Another patient appears to have been affected by agents common to both groups but will be described with the second.

CASE 1.—J. B., a 45-year-old man, gave a five-year history of severe leg cramps, which were due to marked narrowing of the abdominal aorta just above the bifurcation.

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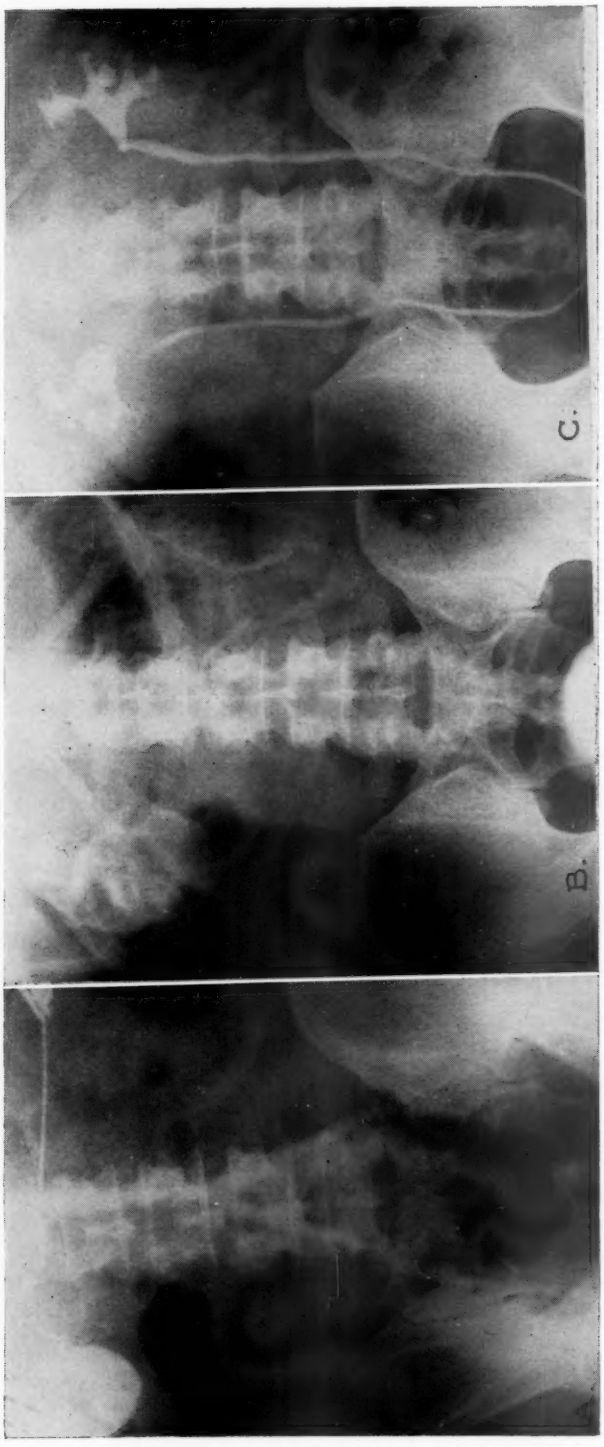

Fig. 1.—*A*, Aortogram. Direct injection into ostium of right renal artery (with gas-pressure machine). *B*, Delayed nephrogram (six hours). Residual dye proves interstitial extravasation; renal swelling. *C*, Retrograde pyelogram (3 months). Atrophy, right renal parenchyma with compensatory left hypertrophy.

On Feb. 26, 1952, translumbar aortography was carried out by the vascular surgeon under intravenous Pentothal anesthesia using 50 c.c. of 70 per cent Diodrast injected with a gas-driven machine which gives a pressure of 70 pounds per square inch. X-ray films showed that most of the dye was injected directly into the right renal artery (Fig. 1, *A*). A few hours later, the patient began to have rather severe right flank and costovertebral angle pain and tenderness. An x-ray film (Fig. 1, *B*) showed that an intense nephrogram was still present on the right. The patient developed nausea and vomiting, with fever to 103° F. The urine was grossly bloody.

The next day, retrograde study with No. 6 ureteral catheters in each renal pelvis demonstrated the following differential function:

Intravenous phenolsulfonphthalein	Right (injured)	Left (normal)
Appearance time	12 min.	2½ min.
15 min.	10 c.c. trace	15 c.c. 17.5%
Specific gravity	1.012	1.028

The patient continued to have nausea and vomiting and mild right flank pain and fever for three more days and then all of the symptoms subsided. He was discharged from the hospital on the eighth day postaortography. Total P.S.P. excretion at this time was 35 per cent in thirty minutes.

Four months later, retrograde urologic study was repeated:

Intravenous phenolsulfonphthalein	Right	Left
Appearance time	12 min.	2½ min.
15 min.	10 c.c. trace	15 c.c. 25.0%
Urine chloride	1.8 Gm./L. (31 meq./L.)	5.9 Gm./L. (100 meq./L.)
Gross appearance	Watery	Bright yellow
pH	6.5 - 7	5.5 - 6
Special gravity	1.012	1.018

Pyelograms (Fig. 1, *C*) at this time showed some atrophy of the right kidney with fairly normal calyceal pattern, and with compensatory hypertrophy on the left. No renal difficulties appeared after a successful thromboendarterectomy of the distal abdominal aorta.

CASE 2.—B. M., a 53-year-old male patient, had a two-year history of severe leg cramps. Aortography was carried out on Dec. 31, 1952, under general anesthesia, with local procaine infiltration, using 50 c.c. of 70 per cent Diodrast injected with the gas-pressure machine. Aortograms showed obstruction of the lower abdominal aorta starting just below the renal arteries and becoming complete at the level of the third lumbar vertebra (Fig. 2, *A*). The inferior portion of the right kidney showed dense opacification still present five minutes after injection (Fig. 2, *B*). It appeared that a high proportion of the charge was injected directly into an aberrant artery going from the aorta to the lower pole of the kidney.

The patient had no apparent untoward effects from this injection and two days later underwent an extensive thromboendarterectomy. No follow-up studies on the right kidney are available, but the patient has had no further symptoms.

CASE 3.—L. C., a 47-year-old man, had teeth extracted on July 24, 1952, and that evening developed acute pain in the left lower leg and foot with numbness and cyanosis, for which two lumbar sympathetic blocks were made. Blood pressure at that time was 170/88. Five days later, translumbar aortography was carried out with a two-needle technique; 70 c.c. of 70 per cent Diodrast was injected. Aortography revealed that one of the needles was directed toward the lumen of the right renal artery and the other needle was outside

Fig. 2.—*A*, Aortogram. Direct injection into anomalous artery to lower pole of right kidney with interstitial and subcapsular extravasation.
B, Nephrogram. Persistent extravasation.

Fig. 3.—Aortogram. Periaortic extravasation and direct injection into right renal artery, resulting in temporary nonfunctioning kidney with hypertension.

the lumen of the aorta (Fig. 3). The patient thus had excessive injection into the right kidney, with extravasation around the renal artery. The arterial supply on the right appeared normal.

A few days following this procedure, the patient's blood pressure increased to 220/110. Three weeks later, bilateral iliac endarterectomy and lumbar sympathectomy were performed. In ten days, the patient began to manifest signs and symptoms of severe hypertensive retinopathy. The blood pressure had risen to 230/120. Seven weeks after aortography, intravenous urograms showed no function of the right kidney with normal function of the left. Retrograde study showed a trace of P.S.P. excretion in fifteen minutes from the right kidney, 25 per cent excretion on the left, with pyelograms demonstrating normal renal outlines.

The patient was followed without further therapy, and within the next six weeks his symptoms disappeared, the blood pressure returned to 140/90 mm. Hg, and intravenous urograms were normal, with good function on the right.

In these three cases, renal damage resulted from direct trauma at the time of aortography. Not only was the needle misdirected in each instance, but the complicating factors of excessive injection pressure, large volumes of dye, and possible irritation by the media were also present. Pressure and volume are interrelated factors, with the medium causing damage by being forced out of the capillaries or by direct irritation because of high concentration. As a result, one patient developed an atrophic kidney, and a second had a temporarily nonfunctioning kidney with severe hypertension which spontaneously reverted to normal within three months. High aortic block is particularly likely to dam back dye and promote renal damage.

II. The remaining four patients fall into the indirect classification. In each of them, the injury was bilateral.

CASE 4.—H. V., a 45-year-old man, had intermittent claudication due to almost complete obstruction of the aorta just below the renal arteries. An aortogram, under spinal anesthesia, was made on Jan. 3, 1952 (Fig. 4), after a previous attempt two and one-half months before had shown merely the mesenteric vessels. A total of 100 c.c. of 70 per cent Diodrast in two divided portions of 50 c.c. each was injected. The patient developed severe headache and a transient urticarial reaction after the first injection. He was given 50 mg. of Benadryl and 100 mg. of Demerol intravenously with relief of the urticaria, but not of the headache. The second injection was accompanied by severe back and abdominal pain, followed by nausea and vomiting. Blood pressure, which was initially 130/80 mm. Hg, rose to 240/90. It remained elevated for thirty minutes, then dropped to 80/50, where it remained for a few hours. It began to rise slowly after a blood transfusion was started. The patient was completely anuric for two days and then became oliguric with twenty-four-hour output varying between 125 c.c. and 470 c.c. A diagnosis of lower nephron nephrosis was made. By the tenth day, the nonprotein nitrogen had risen to 161 mg. per 100 c.c. The patient was treated by careful balance of fluid and electrolytes and of body weight.[4] On the eleventh day, diuresis began and the patient went on from there to complete recovery. On the twenty-second day, the P.S.P. excretion, which had been less than 1 per cent during the period of lower nephron nephrosis, was 24 per cent in one hour. Eight months later, P.S.P. excretion was 65 per cent in one hour, and a successful thromboendarterectomy was performed. The patient had an uneventful postoperative course with no renal difficulties. Of interest is the fact that impotency present before surgery was completely relieved.

CASE 5.—T. C., a 64-year-old man, gave a three-year history of pain, numbness, and tingling in both legs. Aortography was carried out on July 2, 1952 (Fig. 5), under intravenous Pentothal and Demerol anesthesia, after sensitivity test to Diodrast showed no reaction. A total of 60 c.c. of 70 per cent Diodrast was injected through two needles.

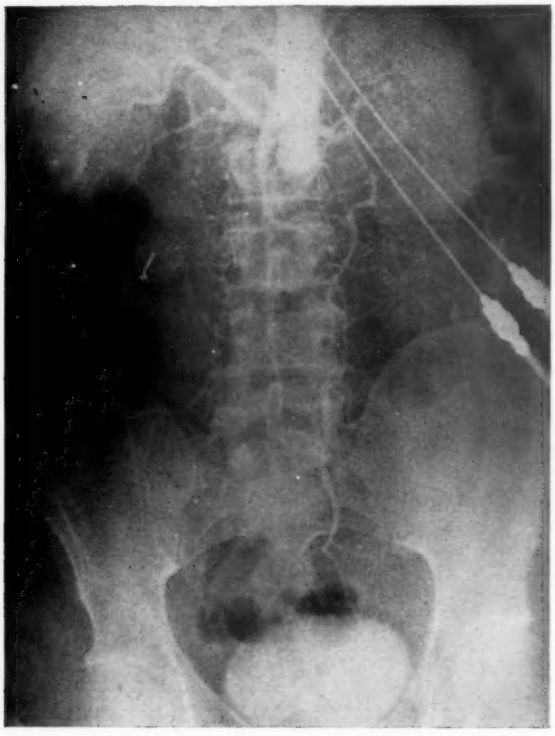

Fig. 4.—Aortogram. Large injection (50 c.c. twice) above high aortic block, resulting in lower nephron nephrosis.

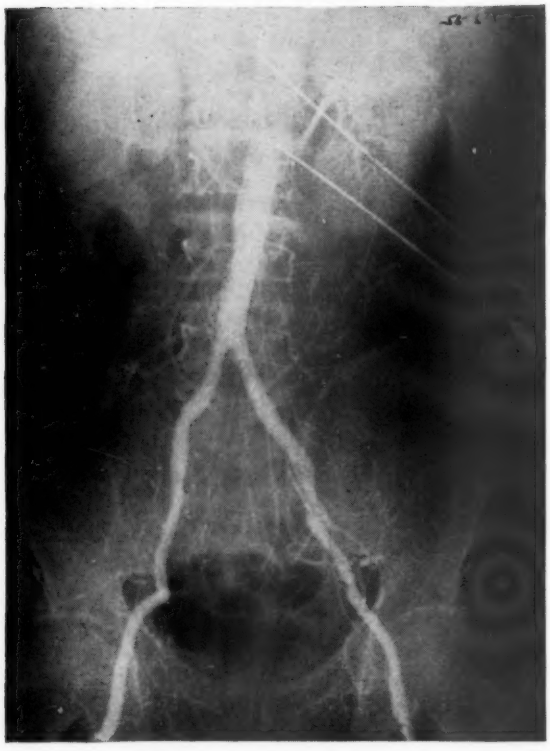

Fig. 5.—Aortogram. Patient developed I_2 sensitivity with rash and rhinorrhea, and lower nephron nephrosis.

On his way home the next day, the patient began to have severe nausea and vomiting, so that four days later he was hospitalized near his home. Examination showed a severe iodide reaction with rhinorrhea, hoarseness, and a typical eruption over the skin of the face, neck, and chest. Urinary output was scant, and N.P.N. was 101 mg. per 100 c.c. He then became anuric and the N.P.N. rose further. Hiccups and vomiting were intractable. He was treated by balance of fluids and electrolytes, and by the tenth day postaortography he began to put out urine in large amounts, with a gradual return of renal function toward normal. One month later, N.P.N. was 33 mg. per 100 c.c. and P.S.P. excretion 45 per cent in 10 minutes.

CASE 6.—N. H, a 60-year-old female patient, had a two-year history of left leg cramps due to narrowing of the aorta starting below the level of the renal vessels and marked obstruction of the right common iliac artery.

Fig. 6.—Aortogram. Large injection into aorta above stenosis, resulting in lower nephron nephrosis.

Aortography was carried out in Dec. 30, 1952, under Pentothal anesthesia with 70 c.c. of 70 per cent Diodrast, using the two-needle technique (Fig. 6). The patient was discharged from the hospital the following day, but three days later was admitted to another hospital near her home because of severe vomiting and back pain which had started twenty-four hours after the aortogram. She was found to be markedly oliguric, putting out 30 c.c. of urine in twenty-four hours. She was treated conservatively and remained oliguric until the eleventh day, when diuresis began. The N.P.N., which had risen to 111 mg. per 100 c.c., fell by the twentieth day to 28.

CASE 7.—L. M., a 58-year-old man, had an abdominal aneurysm and arteriosclerotic constriction of the iliac arteries. On Feb. 13, 1953, translumbar aortography was carried out under Pentothal anesthesia with 50 c.c. of 70 per cent Diodrast injected through a single needle with the pressure machine. (Fig. 7.) There was an intense nephrogram at eight seconds. No immediate untoward effects were noted and there was no evidence of iodine sensitivity. The patient was discharged the following morning, but that afternoon had continuous hiccups. The following morning, he noted that the urine looked like water and that his eyes were a little puffy. Three days later, he consulted a local doctor because

his urine output seemed low; asparagus that he had eaten three days before had given no odor to the urine. At that time, he was found to have gained eight pounds, and serum creatinine level was 13 mg. per 100 c.c. Fluids were restricted and salt was withheld. By the tenth day, the urine output increased in amount and the patient made an uneventful recovery.

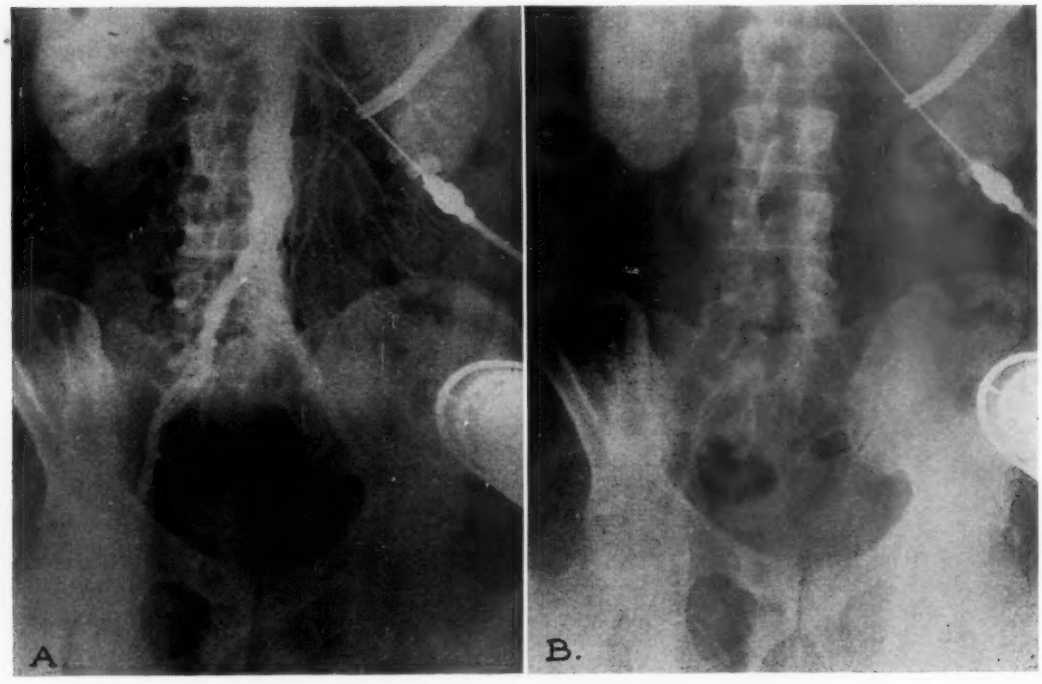

Fig. 7.—*A*, Aortogram. Injection with gas-pressure machine.
B, Nephrogram. Followed by lower nephron nephrosis.

Four patients developed reversible renal failure after aortography (Fig. 8). In three cases, the two-needle technique was used and over 60 c.c. of dye injected. In the other, the gas-pressure machine was used; the patient appeared to have immediate parenchymal damage bilaterally. All recovered completely, after periods of oliguria of ten or eleven days. Two of the four had evidence of Diodrast idiosyncrasy.

That contrast media can cause change in renal function is illustrated in another patient who was subjected to translumbar aortography with the needle placed below the right and above the left renal arteries. Fig. 9, *A* shows the left nephrogram two seconds after injection with no filling of the right kidney, indicating that the dye went only to the left kidney. Twenty-five minutes later (Fig. 9, *B*), the right side showed a normal urogram, whereas the left side showed calyceal spasm and poor filling with persistent nephrogram indicative of at least temporary arrest of the normal tubular transport mechanism.

Experimental Findings.—To test the significance of excessive pressure in terms of renal damage, we performed two types of experiments on dogs.

Type 1. Direct injection of contrast media + India ink into renal artery: After exposure of the dog's left renal artery, 20 c.c. of 70 per cent Diodrast and

5 c.c. of dilute buffered India ink were injected with great force, using a hand-driven lever apparatus[5] directly into the artery. The kidney was immediately removed and perfused. Radiographs showed subcapsular and interstitial extravasation (Fig. 10, *A*), similar to that seen in Cases 1 and 2. The density of the India ink particles in the parenchyma after the kidney was fixed in formalin and cleared by the Spalteholz technique (R. Skarda) is shown in Fig. 10, *B*.

Fig. 8.—Duration of anuria and oliguria in four cases of bilateral injury.

Fig. 9.—*A*, Left nephrogram. Needle inserted below right renal artery.
B, Urogram, twenty-five minutes later. Left nephrogram still present, with calyceal spasm. Normal right urogram.

Type 2. Forceful injection of Diodrast and India ink into the aorta occluded just below the renal arteries:

(A) To study immediate effects, the dog's aorta was exposed transperitoneally and occluded manually above and below the origin of the left renal artery during forceful injection of 20 c.c. of 70 per cent Diodrast and 5 c.c. of dilute buffered India ink. The kidney was removed and perfused and a radiograph was taken. Extravasation into the cortex was evident (Fig. 10, *C*), although less extreme than in the first experiment. After fixation and

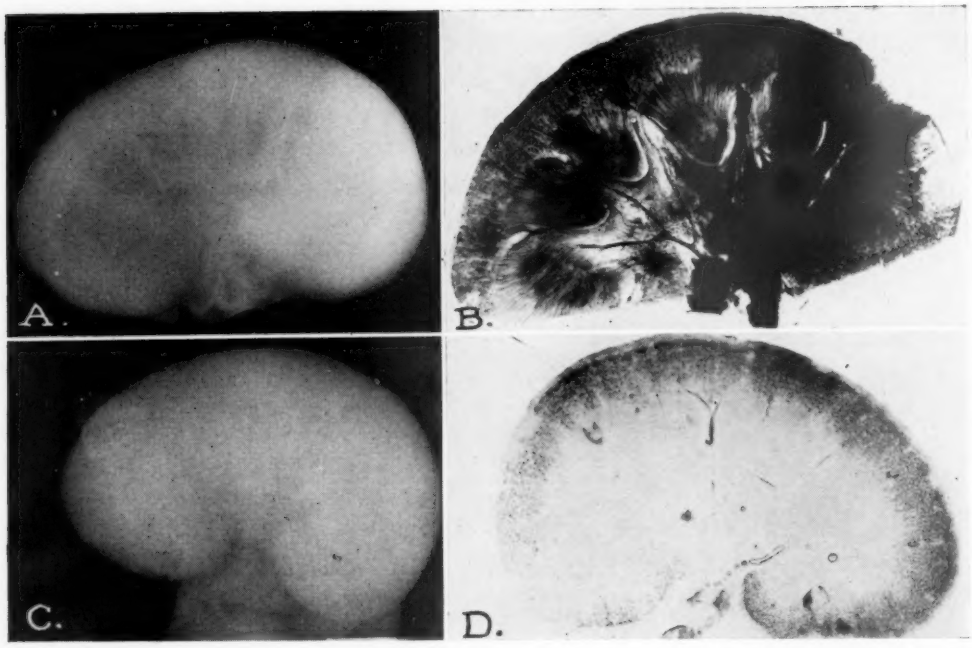

Fig. 10.—*A*, Radiograph of dog kidney showing interstitial and subcapsular extravasation after direct arterial injection of Diodrast and India ink.

B, Cleared sagittal section of same kidney.

C, Radiograph of dog kidney showing interstital extravasation after forceful injection of aorta.

D, Cleared sagittal section of same kidney. Note intercapillary filling of Bowman's space.

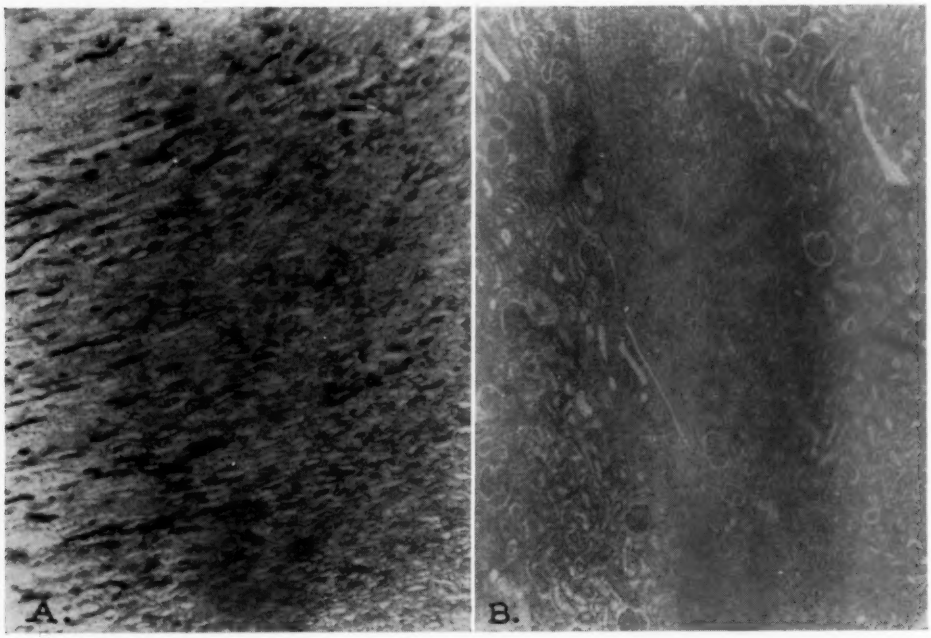

Fig. 11.—*A*, Photomicrograph (×100; reduced ⅕) of dog kidney shown in Fig. 10, *C* and *D*. Particles can be seen in the tubules and interstitially.

B, Photomicrograph (×100; reduced ⅕) of dog kidney removed three weeks after forceful injection. Note patchy interstitial fibrosis.

clearing, the kidney still showed extravasation of India ink in Bowman's space (intercapillary) and in the tubules and intertubular spaces (Figs. 10, *D* and 11, *A*).

(B) To study the delayed effects, a similar injection was made and the kidney left in situ. At the end of three weeks, microscopic examination showed patchy areas of interstitial fibrosis (Fig. 11, *B*).

Although these experiments could be amplified, they illustrate a way whereby forceful injection of a substance may cause extravasation into the renal parenchyma, which results in renal damage.

DISCUSSION

These clinical and experimental results indicate that renal damage may be caused by high pressures combined with a volume of media sufficient to generate such pressure, or by irritation of the contrast media itself if the pressure-volume relationship produces a sufficiently high concentration. The damage appears to come either from mechanical damage to the renal blood vessels with extravasation or from excessively high concentrations of the dye as it passes through the kidney.

To insure against such damage following aortography, certain limitations in technique appear desirable. If the whole aortic tree must be filled (as before vascular surgery), then at least 50 c.c. of dye is needed. To prevent excessive pressure (or the danger of trauma by too large a needle), it has been found advisable to use two smaller 17 or 18 gauge needles and inject by hand with two syringes. To avoid direct injection of the renal vessels (or other aortic branches, for that matter), the needle tips should be angulated sharply upward so that they enter the aorta just below the diaphragm and 2 to 3 cm. above the renal vessels. Long needles are required.

For urologic purposes, such large volumes of media are not needed, although the 12 c.c. volume advocated by Parke Smith[6] has not been enough in many cases to show the renal vascular pattern. We have found 25 c.c. to be adequate. But this quantity of dye injected directly into the renal artery, even by hand pressure, could be damaging, so we advocate, especially for the occasional operator, the making of a scout film after injection of 5 c.c. of dye to determine the position of the tip of the needle. It may then be moved should it lie within the lumen of the renal artery or mesenteric vessels, or should there be extravasation. This precaution might have obviated the complications in at least three of the reported cases.

These smaller volumes used to visualize just a segment of the aorta and the renal vascular tree can be conveniently injected under local anesthesia. The use of local anesthesia avoids some of the dangers inherent in general anesthesia, particularly when given away from the operating rooms, and avoids the need of an anesthetist and additional personnel. The patient is heavily premedicated with barbiturates and intravenous Demerol, and a single 17 gauge needle is inserted by local infiltration of 1 per cent procaine. Once the aorta is entered, an additional 20 c.c. of procaine is injected directly into the aorta to avoid vasospasm in the distal vessels. With 70 per cent Diodrast there has

occasionally been a sudden movement by the patient from the "shock" of the injection, but this was controlled with adhesive taping. Since using 70 per cent Urokon, we have not seen this sudden movement.

SUMMARY

1. Renal damage following translumbar aortography has been observed in seven patients.

2. The direct forceful injection of a sizable amount of dye into a single renal artery produced unilateral damage in three of these patients. The high pressure produced visible extravasation of an irritating medium.

3. Bilateral renal injury followed by reversible renal failure occurred in four other patients. Two of the four had evidence of Diodrast sensitivity; three of the four had at least 60 c.c. of media injected, and the fourth had 50 c.c. injected under high pressure.

4. Animal experiments showed that high injection pressures caused interstitial extravasation of the medium, leading to late renal fibrosis.

5. A safer plan for aortography is proposed, using lower pressures and smaller volumes.

We acknowledge the asistance of Dr. John W. Schulte, who prepared the detailed data on patient No. 1, and we thank him for his interest and his valuable suggestions.

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PROSTATIC CANCER

IX. Value of Transurethral Biopsy in Search of Early Prostatic Carcinoma

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CURRENT trends in cancer therapy are such that the early detection of adenocarcinoma of the prostate gland is receiving renewed interest and attention.[1, 2] The inadequacy of rectal palpation diagnosis has been demonstrated, particularly in regard to the detection of malignancies at a stage in their development preceding spread from the original focus to other parts of the body.[3] Tissue diagnosis assumes greater importance in the absence of definitive points in the medical history or of diagnostic signs which are suggestive of prostatic cancer.

Three methods for obtaining tissue from the prostate gland are utilized to detect curable cancer. Cytologic study of the expressed prostatic secretion smear is the newest of these techniques.[4] A second method involves the use of a needle[5] or punch instrument[6] which is introduced through a small perineal stab incision to obtain a core of posterior prostatic tissue. Open perineal dissection for direct wedge biopsy of the posterior lamella is the third method.[7, 8]

There has been no experience with the prostatic smear technique in this institution. Objections to the method center about the inadvisability of massaging adenocarcinomas, which are known to spread in perineural lymphatic spaces, as well as the statement by Peters and her associates[4] that there is a definite percentage of false positive impressions of cancer which are gained from a series of examinations of this sort. In addition, Colby[9] has found that only 40 per cent of his series of 100 cases of operable prostatic cancer yielded a positive diagnosis from the prostatic smear examination. There is the further theoretical objection that early prostatic cancer often may not have an anatomic connection with the ducts emptying into the prostatic urethra, thereby making it impossible to express cancer cells from a small focus in the posterior prostate. While the punch biopsy technique is useful in documenting cases of more advanced carcinoma, it does not compare favorably with the open surgical biopsy technique in cases of early prostatic cancer.[10] Open perineal prostatic biopsy appears, then, to be the most reliable method for detecting early prostatic cancer, which is thought to develop in 85 per cent of such malignancies in the posterior portion of the gland.[11-14]

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It is mainly in an attempt to detect the 15 per cent of early carcinomas which originate in the periurethral portion of the prostate gland that transurethral prostatic biopsy has been employed.

CLINICAL STUDIES

During an evaluation of over 300 men by various methods and techniques designed to detect early prostatic cancer,[15] a transurethral prostatic biopsy has been included in the study for fifty-nine consecutive patients. In each instance, open surgical perineal prostatic biopsy was performed concurrently with transurethral biopsy.

The technique for transurethral prostatic biopsy is as follows: After anesthesia is instituted, a No. 28 Fr. Stern-McCarthy electrotome sheath is introduced through the urethra. Three areas of the prostate are routinely sampled, namely, the floor of the prostatic urethra and a mid-section of each lateral prostatic lobe. Specimens measure approximately 2 cm. in length and from 0.5 to 1.0 cm. in depth. Whenever a median lobe enlargement is encountered, a central core of median tissue is excised. Bleeding is controlled by electrocoagulation. The entire amount of tissue is removed within five minutes, obviously involving less time and tissue than in a therapeutic resection.

The specimens are fixed in Bouin's solution, made into paraffin blocks, sectioned at 5 microns, stained with hematoxylin and eosin, and studied microscopically.

The surgical pathologic material upon which evaluation of transurethral prostatic biopsy is based consists of fifty-nine consecutive transurethral prostatic biopsies, fifty-nine concurrently obtained open perineal biopsies of the posterior prostate, and forty-five final surgical specimens obtained from definitive surgical procedures upon the prostates of these patients.

RESULTS

Among the fifty-nine patients evaluated, there were forty-three in whom a benign tissue diagnosis was finally established. Of these, all forty-three were correctly diagnosed by both the open perineal biopsy and by the transurethral biopsy techniques. In the case of carcinoma, there were sixteen positive cases revealed by final pathologic diagnosis upon surgical specimens. Of the sixteen, eight cases, or 50 per cent, were unsuspected carcinoma and

TABLE I. ANALYSIS OF FIFTY-NINE TRANSURETHRAL RESECTION AND PERINEAL PROSTATIC BIOPSIES

Number of cases biopsied by both routes	59
Number of benign lesions	43
1. Correctly diagnosed by transurethral resection biopsy	43
2. Correctly diagnosed by perineal biopsy	43
Number of prostatic cancers	16
1. Correctly diagnosed by transurethral resection biopsy	8
2. Correctly diagnosed by perineal biopsy	8*

*The eight cases of prostatic cancer detected by transurethral resection biopsy were in known metastatic instances. The prostate was biopsied only for tissue confirmation of the clinical diagnosis.

eight were cases in which metastases were known to exist prior to the biopsy. All sixteen of the cancers were revealed by open perineal biopsy method. Of the eight cases of advanced cancer, the transurethral biopsy was positive. Of the eight early unsuspected carcinomas, none yielded a positive transurethral biopsy finding (Table I).

DISCUSSION

The results of this investigation suggest that little likelihood exists of diagnosing early prostatic cancer with a resectoscope. A more extensive resection than the one utilized might yield a higher diagnostic rate for malignancy. However, there are objections to extensive transurethral resections for this purpose. The fact that irrigating fluid almost always enters the blood stream during extended prostatic resection is well established.[16] It is probable that prostatic cancer cells are introduced into the blood stream in precisely the same way. In addition, extensive resection would "make the prostatic remnant unsuitable for total perineal extirpation unless it were done within 24 hours."[17]

It is probable that tumors detected during examination of transurethral prostatic curettings are those which originate in the posterior prostate, and then grow toward the urethra to a point at which they have invaded periurethral glandular hyperplasia. Such tumors are beside the subject under consideration, namely, the detection of early prostatic cancer. Suffice it to state that the resectoscope is useful for the documentation of advanced prostatic cancer when a tissue diagnosis has not previously been obtained, but with the clear knowledge of the risk of tumor spread in mind.

It should be emphasized that the modern electrotome does not alter the morphology of the specimens in such a way that distortion is a prime factor in hindering accurate microscopic appraisal.

Other studies from this institution have indicated that clinical accuracy for the detection of early prostatic cancer by the open perineal prostatic biopsy method is 89 per cent.[2, 3, 8, 10, 15] The results cited in this communication indicate that transurethral biopsy will rarely, if ever, yield a positive diagnosis of prostatic cancer while the disease is in its earlier phases of growth. Because it is technically dangerous, if not impossible, to include posterior prostatic tissue in a transurethral resection biopsy, the likelihood of missing posterior prostatic cancer—the commonest form—is extremely high.

The superiority of open perineal prostatic biopsy over transurethral biopsy seems definite. The discrepancy between the diagnostic values of the two techniques is so great that the transurethral method may reasonably be abandoned as a device for establishing the tissue diagnosis of unsuspected early prostatic carcinoma.

SUMMARY

1. A series of fifty-nine consecutive patients subjected to concurrent transurethral and open perineal surgical biopsy of the prostate is presented.

2. Sixteen adenocarcinomas were found, eight of which were known metastatic cases, and eight of which were totally unsuspected malignancies.

3. Transurethral biopsy yielded a diagnosis of cancer only in the eight known metastatic cases.

4. Transurethral biopsy yielded an erroneous diagnosis of benign prostatic tissue in all eight of the early, unsuspected cancers.

5. All of the sixteen cancers were detected by open perineal biopsy.

6. Transurethral biopsy may reasonably be abandoned as a means of detecting early prostatic cancer.

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MIXED TUMORS IN UNUSUAL SITES

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MIXED tumors of anlage origin, the so-called mixed tumors of the salivary glands, have a structure mimicking an organ. They usually occur in or near the parotid or submandibular glands, and occasionally in or near the sublingual glands, or elsewhere along the lines of embryonal facial fissures. In rare instances, they have been observed in parts distant from the head. The frequency with which these mixed tumors occur in unusual sites is as yet undetermined. Information is accumulating, however, revealing that they occur more often than was formerly believed. In this paper, six such tumors are presented, one in each of the following locations: the scalp, the lower eyelid, the hard palate, the lower lip, the back, and the testis. That on the back is believed to be the second such growth recorded in this location. The growth in the testis is included even though other observers may interpret it as a teratoma.

CASE REPORTS

CASE 1.—A. M. C., a 58-year-old white man, was admitted to the Veterans Administration Hospital, Houston, Texas, July 6, 1953, for treatment of painful varicose veins. On examination a freely movable, slightly tender mass, 2 cm. in diameter, was noted in the right temporal region just above the auricle. The patient said that the mass had been present for many years, that it seemed to vary in size, and was sometimes tender, but never painful. Following removal, the wound healed and there was no recurrence.

The specimen (S-1331-53) consisted of a thin-walled multiloculated sac, 1.5 by 1.5 by 0.6 cm., the locules containing a tan transparent coagulum. Microscopically, various-sized spaces contained a pale pink coagulum and were lined with low columnar cells, with the nuclei heaped into several rows. The septa were delicate, with occasional projections. Some of these had a stroma of embryonal connective tissue partly hyalinized. In it were acinar tubular structures lined by cuboidal cells and endothelial-lined slitlike spaces (Fig. 1).

CASE 2.—A. R. T., a 53-year-old white man, was admitted to the Veterans Administration Hospital, Houston, Texas, Nov. 15, 1949, with a mass on the left lower eyelid of about five years' duration. It had developed slowly, and during the previous year there had been mild conjunctivitis. At the time of admission, the mass was 1 cm. medial to the lateral canthus and was 0.3 cm. in diameter. Following removal, the wound healed promptly, and there has been no recurrence to date.

The specimen (S-764-49) consisted of a wedge of tissue, 0.5 by 0.3 cm., partly covered with skin, that had a raised area 0.2 cm. wide. Microscopically, in the corium, beneath a delicate layer of stratified squamous epithelium, there

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was a fairly well-circumscribed field of strands of epithelial cells that enclosed various-sized lumina, empty or filled with pink amorphous substance. The cell strands had a lacelike pattern, with palisading at the periphery. There were lobules of adipose tissue cells within the loose connective tissue stroma (Fig. 2).

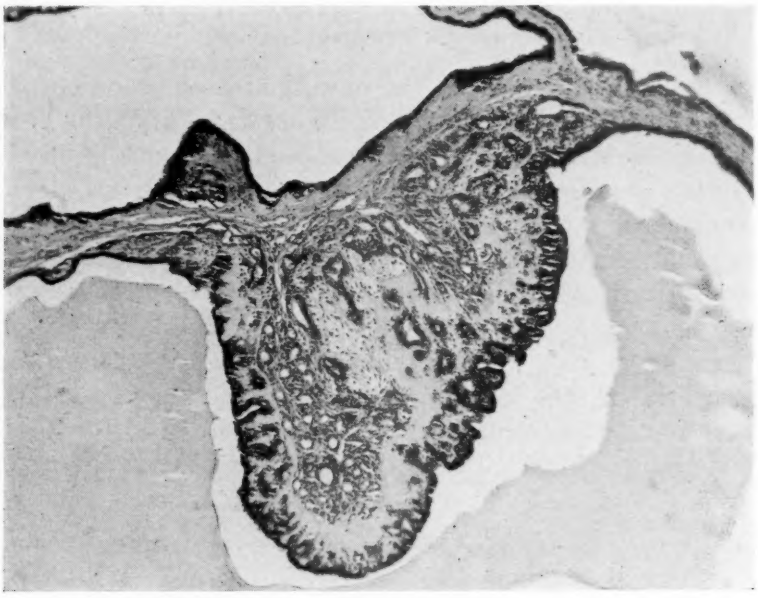

Fig. 1.—Mixed tumor from the right temporal region of a man aged 58 years (Case 1). In the stroma are stellate embryonal connective tissue cells. (×85.)

Fig. 2.—Mixed tumor from the left lower eyelid of a man aged 53 years (Case 2). In the stroma are scattered adult adipose tissue cells. (×80.)

CASE 3.—C. L. J., a 58-year-old white man, was admitted to the Veterans Administration Hospital, March 13, 1952, with a painless tumor of the hard palate for eight years. At the time of admission there was a firm bulging mass, 4 by 3 cm., in the hard palate on the left that extended posteriorly for 1 cm. into the soft palate (Fig. 3). The mass was excised and found to be limited to the soft tissues of the palate. Following operation, a naso-oral fistula developed that healed spontaneously. There has been no recurrence to date.

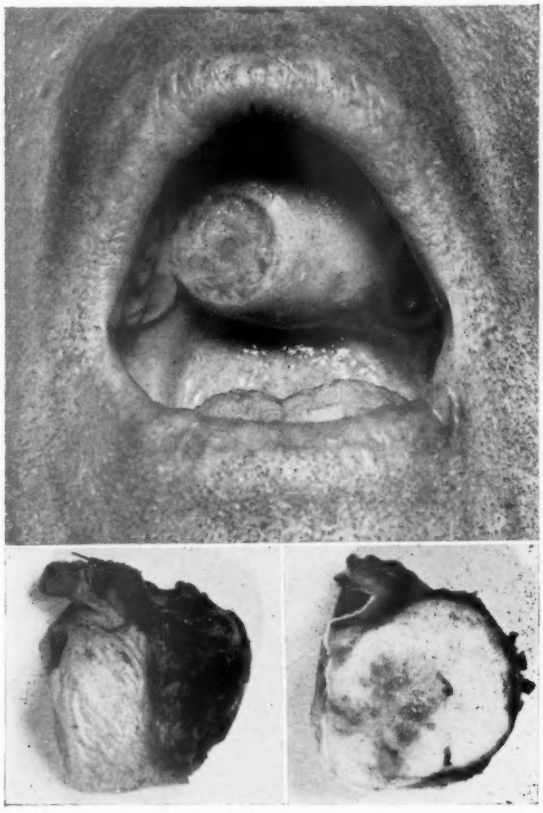

Fig. 3.—Mixed tumor in the palate of a man aged 58 years (Case 3). The growth is seen in situ, and the external and cut surfaces of the removed specimen are also shown.

The specimen (S-491-52) consisted of a globular mass of firm tissue, 3.5 by 3 by 3 cm. One surface was partly covered by mucous membrane eroded in an area measuring 1.5 cm. The cut surfaces were yellow-gray, mottled with red (Fig. 3). Microscopically, strands and nests of epithelial cells, some in a concentric pattern with central keratinization and frayed borders, were within a loose or more dense and hyalinizing connective tissue stroma. In the latter, lobules of adult adipose tissue cells were conspicuous (Fig. 4).

CASE 4.—G. C. S., a 62-year-old white man, was admitted to the Veterans Administration Hospital, May 28, 1952, for arthritis. On examination, a nodule, 1 cm. in diameter, was noted on the inner surface of the left lower lip, which was said to have been present for six years. At the time of excision it appeared encapsulated. When the patient was last seen, May, 1953, there had been no recurrence.

The specimen (S-987-52) consisted of a globular portion of tissue, 1 by 0.8 by 0.4 cm., partly covered by mucous membrane, measuring 2 by 0.3 cm.

The cut surfaces revealed a solid nodule within a delicate capsule. Microscopically, there was a well-circumscribed area of compactly arranged, varying sized epithelial cells in a streamlike pattern. The cells had vesicular nuclei

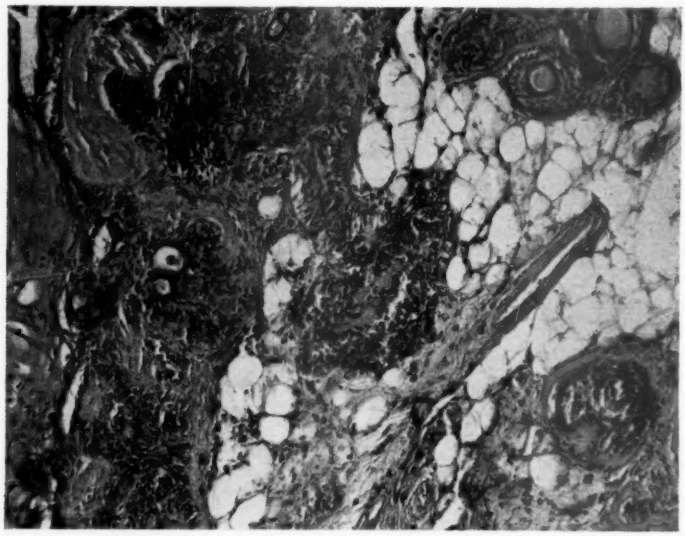

Fig. 4.—Microscopic structure of the growth shown in Fig. 3. The adult adipose tissue cells in the stroma are believed to be an intrinsic part of the neoplasm. (×75.)

Fig. 5.—Mixed tumor from the lower lip of a man aged 62 years (Case 4). The compactly arranged epithelial cells have a streamlike pattern. Elsewhere cuboidal cells line acinar or elongated spaces. (×80.)

in abundant pink or lavender cytoplasm. Elsewhere cuboidal cells lined minute lumina or elongated spaces. The connective tissue stroma was scanty in places and elsewhere abundant, dense, and hyalinizing (Fig. 5).

CASE 5.—J. C. B., a 62-year-old white man, was admitted to the Veterans Administration Hospital, March 27, 1951, with osteoarthritic pain. On examination, a tumor mass,

10 by 5 cm., was discovered in the left lumbar region. The mass was under the skin, freely movable, and slightly tender. The patient stated that a similar mass was removed from his back during World War I. He believed that the growth recurred in the same region about three years later. It had enlarged slowly during the previous thirty years. The growth was excised, the wound healed, and when the patient was seen one year later there had been no recurrence.

Fig. 6.—Mixed tumor from the left lumbar region of a man aged 62 years (Case 5). Cystic spaces filled with dark liquid are seen on the cut surface of the removed specimen.

The specimen (S-650-51) consisted of a firm mass, 4 by 2.8 cm., attached to an ellipse of white skin, 7 by 3 cm. On section, shiny brown and yellow tissue contained cystic spaces filled with dark liquid (Fig. 6). Microscopically, the neoplastic area was deep in the corium beneath the level of hair follicles and sweat glands. It was delimited by a fairly dense hyalinized fibrous connective tissue capsule. Inside the capsule there were acinar tubular structures lined by low cuboidal cells. There were also large spaces lined by flat cells and filled by pink coagulum. Some of these contained many large mononuclear cells with foamy cytoplasm. These epithelial cell groups were in a scanty loose or dense connective tissue stroma, with occasional broad bands of hyalinized connective tissue. Elsewhere, in loose fibrillar tissue, epithelial cell nests in an acinar pattern faded into connective tissue. Interspersed were fields of young connective tissue with a loose streamlike arrangement of the cells and fibrils (Fig. 7).

Case 6.—A. J., a 42-year-old white man, was admitted to the Psychiatric Service of the Veterans Administration Hospital, March 14, 1952. On physical examination a small nodule was noted in the right testicle. The patient had discovered the nodule about six weeks prior to admission. At operation, a nodule about 1 cm. in diameter was removed from inside the tunica albuginea of the right testicle. Several months following excision there had been no recurrence of the growth.

The specimen (S-548-52) consisted of a portion of gray-white tissue, 1.2 by 1.0 by 0.8 cm. Microscopically, nests and strands of epithelial cells with deep blue nuclei and hardly any cytoplasm were unevenly distributed in a pale fibrillar and vacuolated ground substance (Fig. 8). At one margin there were a few seminiferous tubules which were lined by Sertoli cells and contained no sperms.

COMMENT

The neoplasms herein recorded are essentially similar in their microscopic structure. While they resemble closely those arising in and about the salivary glands, they are of less complex pattern, and the epithelial elements usually

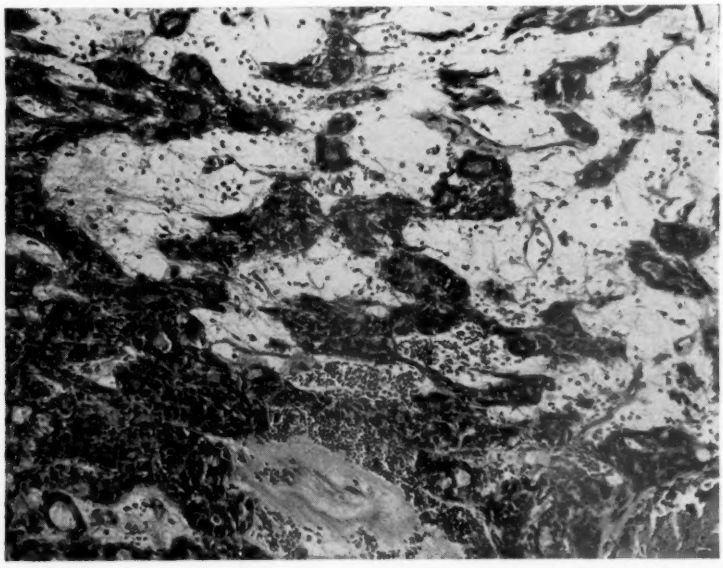

Fig. 7.—Microscopic structure of the growth shown in Fig. 6. In a loose fibrillar substance epithelial cell nests in an acinar pattern fade into connective tissue. (×80.)

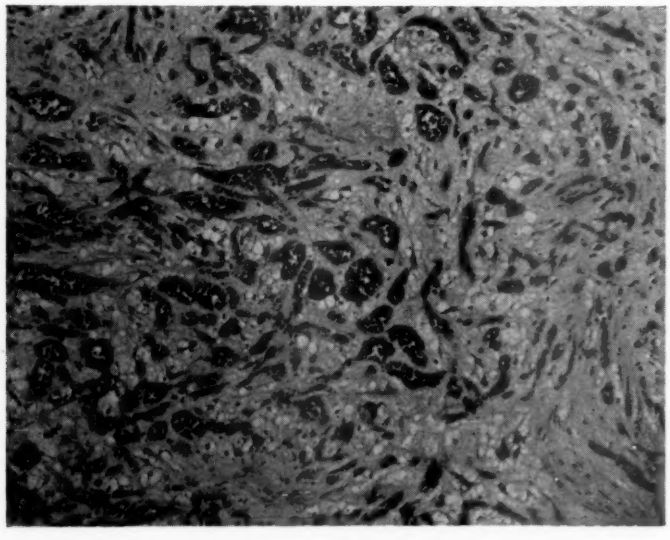

Fig. 8.—Mixed tumor from the right testicle of a man aged 42 years (Case 6). Nests and strands of epithelial cells are unevenly distributed in a pale fibrillar and vacuolated ground substance. (×75.)

predominate. These mixed tumors are fairly common about the oral cavity and the face, at some distance from the large salivary glands. They are more often observed in the palate[1-6] and in the upper lip[7] than elsewhere about the

oral cavity. They have been reported about the nose,[8] on the lower lip,[9] on the eyelids,[8, 10] on the cheek,[8, 11] on the eyebrows,[8, 10] and, less frequently, in the tongue,[12] the tonsillar fossa,[13] larynx,[14] the pterygomaxillary space,[15] and the nasal septum.[16] They have been observed also on the scalp,[17, 18] on the ear,[19, 20] on the postauricular region, and on the neck.[17] Among those located distant from the head, fourteen were recorded as occurring on the upper extremities,[8, 10, 20, 21] eight on the lower extremities,[8] two each in the mammary gland and over the sternum,[10] and one each on the back, inguinal region, vulva,[10] vagina,[22] and buttock.[8]

An understanding of the morphologic features and clinical behavior of these neoplasms is of utmost importance in the approach to a rational therapy.[23] The close similarity in their microscopic structure and behavior suggests a common derivation. It has been inferred that they are derived of ectoderm, the anlage having separated at a stage when the cells as yet had not acquired their dominant characteristics as ectodermal or mesodermal cells.[24] The proportion of ectodermal and mesodermal elements may vary considerably, with at times the mesodermal elements predominating. The testicle itself is a mesodermal structure, hence both the epithelial and nonepithelial elements of the growth are likewise derived of mesoderm. Placing this growth into the group of mixed tumors of anlage origin is therefore not inconsistent with the previously proposed theory of their origin.[24]

SUMMARY

Mixed tumors of anlage origin, the so-called mixed tumors of salivary gland type, occasionally occur on the face and head where salivary glands are not usually present. They have also been observed at sites distant from the head. Observations are recorded on six such tumors, one each of the following locations: the scalp, the lower eyelid, the hard palate, the lower lip, the back, and the testis. That on the back is believed to be the second such growth recorded in this location, while that in the testis may also be interpreted as a teratoma.

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A MODIFICATION OF THE HUNTERIAN LABORATORY ANESTHESIA APPARATUS FOR EXPERIMENTAL SURGERY

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HANLON, Johns, and Thomas,[1] in 1950, described an apparatus which is used in the Surgical Hunterian Laboratory of the Johns Hopkins Medical School for providing anesthesia in experimental thoracic surgery. This apparatus consists of a motor-driven valve which periodically interrupts a stream of compressed air or oxygen passing over an ether reservoir into an endotracheal tube. The source of air or oxygen, the ether reservoir (a one-liter Woulfe bottle), and the interrupter valve are interconnected by means of suitable lengths of rubber tubing. The concentration of ether vapor in the anesthetic mixture may be varied by partially diverting the stream of air which passes over the ether fluid. This is accomplished by the application of one or two hand clamps at particular points on the rubber tubing which connects the ether reservoir to the other units of the system (Fig. 1).

For the past two years we have used a modified form of this apparatus in the Experimental Surgical Laboratory of the University of California Medical Center at Los Angeles. It has provided satisfactory anesthesia for use in both thoracic and prolonged abdominal procedures. The modifications which we have imposed upon the original model have proved to be of sufficient value to warrant their description. These modifications consist of: (1) an air reservoir which is interposed in the line between the air or oxygen supply and the ether reservoir (Fig. 2), (2) a multiple foot pedal device, which replaces the hand clamps, for varying the ether concentration (Figs. 2 to 5), and (3) a blowoff valve which eliminates the expired gases from a point just proximal to the endotracheal catheter (Figs. 2 and 6).

Air Reservoir.—As the interrupter valve rotates, it periodically opens and closes the circuit leading into the endotracheal tube (Fig. 1, *3*). Inasmuch as the flow of compressed air or oxygen is constant, the pressure in the system proximal to the interrupter alternately increases and decreases with the closing and opening of the valve. Hanlon and associates[1] noted that this increase in pressure is dissipated by the expansion of the rubber tubing and compression of the vapor in the ether reservoir (Fig. 1, *5*). Elimination of this cushioning effect by completely diverting the Woulfe bottle (clamps applied at *10* and *11*, Fig. 1) may cause respiratory distress or separation of the connections proximal to the clamps. While, admittedly, a minimal amount of ether vapor will flow through the interrupter valve if only one of the two

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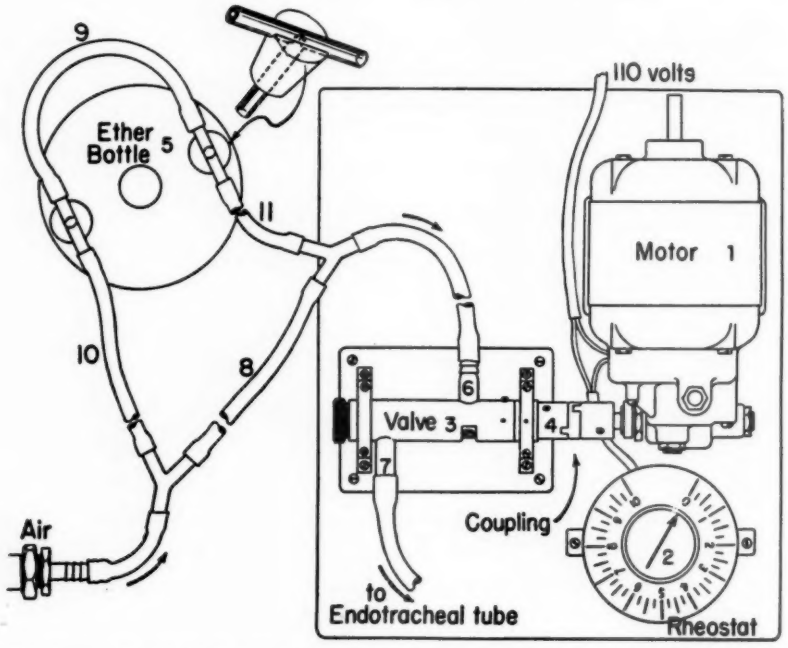


Fig. 1.—Diagram of original apparatus described by Hanlon, Johns, and Thomas.[1] (From Hanlon, Johns, and Thomas.[1])

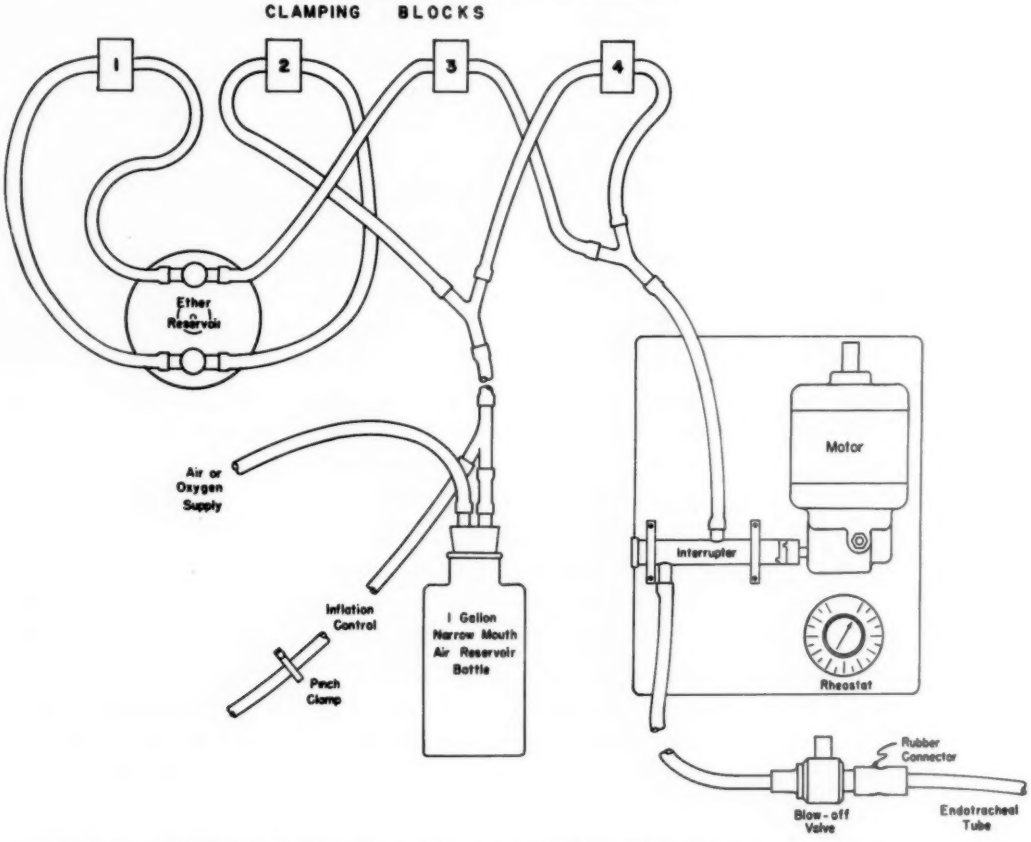


Fig. 2.—Diagram of modified apparatus showing relationships of air reservoir bottle, clamping blocks of foot pedal control mechanism, and blowoff valve. Note lung inflation control in airline.

tubes leading from the Woulfe bottle is occluded, we have found that by interposing an air reservoir (a one-gallon narrow-mouthed bottle) into the system, as indicated in Fig. 2, a separate expansion chamber is provided, so that if the ether reservoir should be completely diverted by compression of both tubes at the same time (at clamping blocks *2* and *3*, Fig. 2), the increase in pressure is absorbed by the compression of air in the reservoir bottle. By this simple device, delivery of ether-free air or oxygen may be accomplished and the apparatus may be used to additional advantage as an air or pure oxygen insufflator or resuscitator.

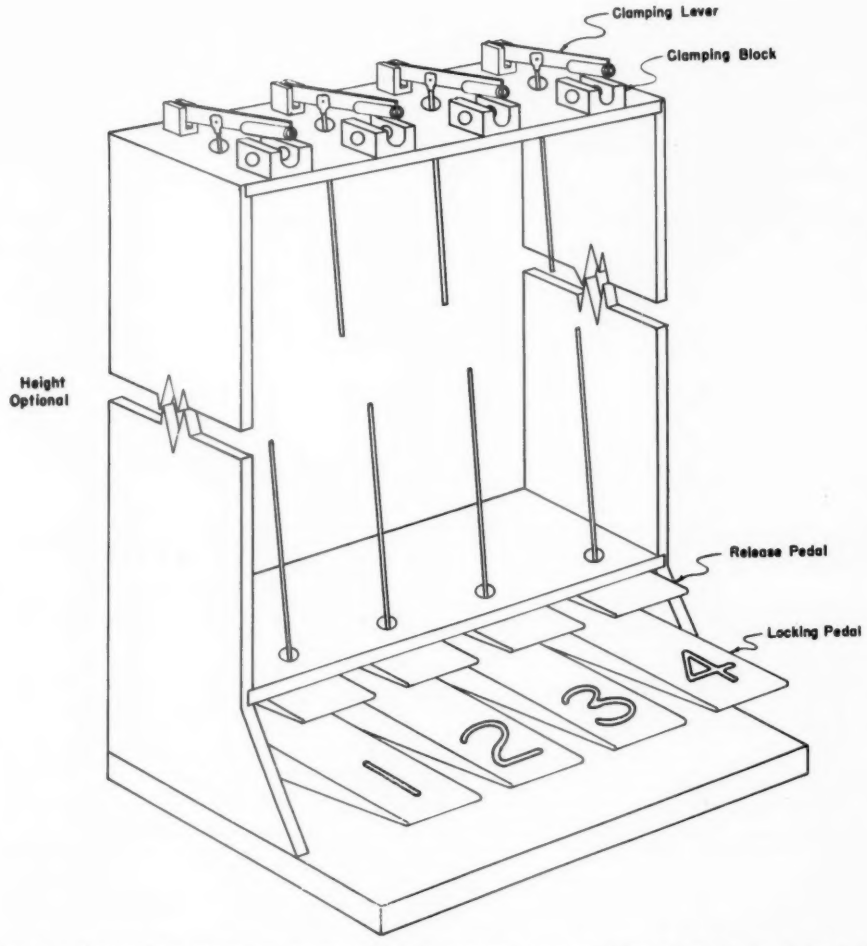
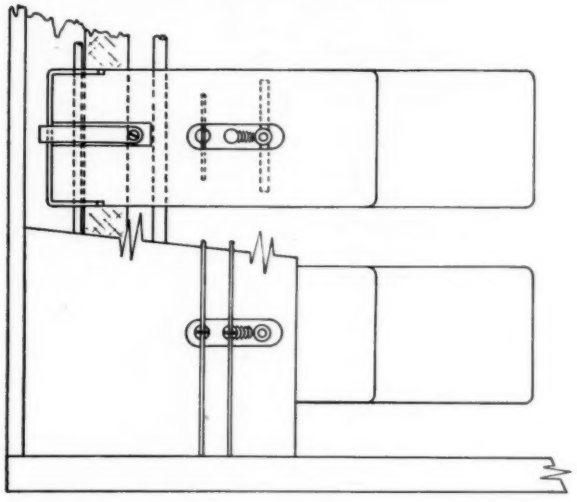


Fig. 3.—Isometric diagram of foot pedal control device (see also Figs. 4 and 5). By pressing down upon any of the four locking pedals, tension is applied to the connecting rod, which in turn lowers the clamping lever. When the foot pedal attains the required pressure (adjustable in the connecting rod), the locking latch is engaged. This maintains the depressed position of the clamping lever until the release pedal is pressed. The release pedal disengages the locking latch, which allows the locking pedal to be pulled up to its original position by the return spring.

In Fig. 2, a safety valve has been inserted in the air line to control the degree of lung inflation. If the tubing and pinch clamp are sterilized and brought into the operative field, this valve may be adjusted by the operator or his assistant as required during the procedure.

Foot Pedal Control.—The addition of a mechanical foot pedal device for the purpose of varying the ether concentration has been the direct outgrowth of a need to conserve the number of unscrubbed assistants engaged in any particular procedure. This innovation allows the surgeon or his assistant, without contaminating himself, to change the concentration of the ether mixture by a simple movement of his foot. The foot pedal control mechanism has been installed at the head of the table directly beneath the anesthesia apparatus. It is constructed in such a way as to permit compression of one or a combination of tubes which connect the ether reservoir to the other units of the system in exactly the same manner as would ordinarily be accomplished by the hand clamps. A diagram of the apparatus with details of the working parts is represented in Figs. 3, 4, and 5.



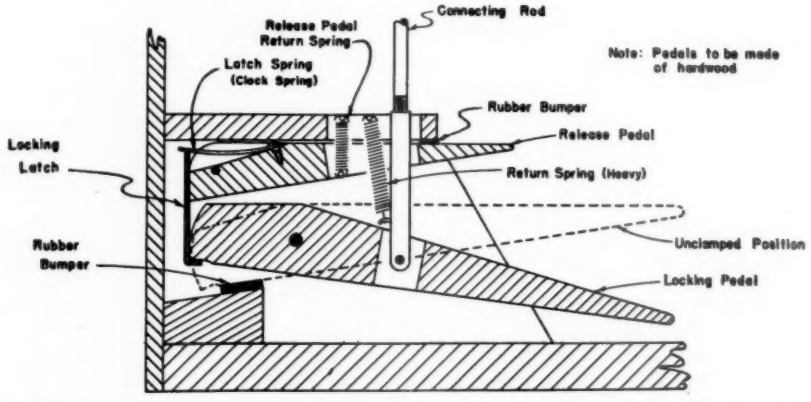
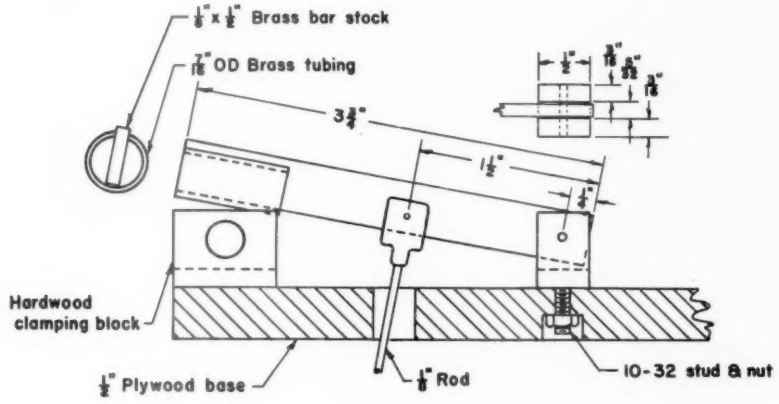


Fig. 4.—Projection drawing in two views showing locking and release pedal details of foot pedal control device (see legend, Fig. 3).

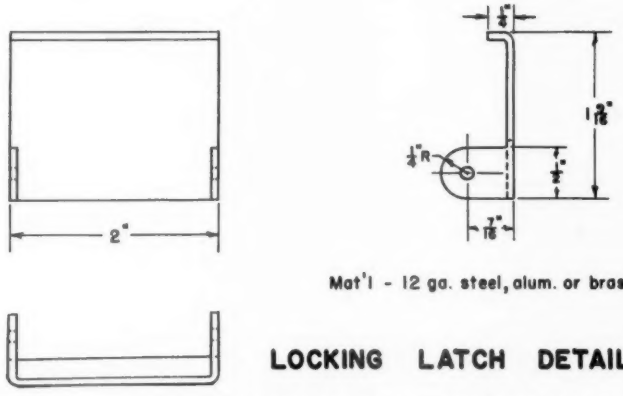
The concentration of ether vapor in the anesthetic mixture may be varied by engaging the four locking pedals of the controlling device singly or in combination, as follows:

Pedals 2 and 3	Ether-free air or oxygen
Pedals 2 or 3	Minimal ether vapor
All pedals released	Moderate ether vapor (usual maintenance level)
Pedals 1 or 4	Heavy ether vapor
Pedals 1 and 4	Maximal anesthetic effect

Blowoff Valve.—The interrupter valve allows for exhaustion of expired gases with each revolution of its cylinder from an opening immediately opposite the intake port (Fig. 1, *6*). The greater the length of tubing which conducts the ether mixture from the apparatus to the animal, the greater the volume of dead space in the system. To avoid this problem, we have inserted



CLAMPING LEVER DETAILS



LOCKING LATCH DETAILS

Fig. 5.—Drawing showing clamping lever and locking latch details of foot pedal control device (see legend, Fig. 3).

a simple blowoff valve of our own design just proximal to the endotracheal catheter. This valve is similar in principle to those used by clinical anesthetists. While it is the most recent of the innovations which we have applied to the apparatus, the blowoff valve has already proved to be of considerable value in controlling the amount of carbon dioxide which the animal is required to rebreathe. A detailed drawing of this valve is seen in Fig. 6.

DISCUSSION

It has been our practice to induce anesthesia, after suitable medication with morphine and atropine, with an intravenous injection of 2½ per cent thiopental sodium or parenteral pentobarbital sodium. We have found this to be much simpler than the use of open drop ether. After the endotracheal catheter is passed and the cuff inflated, connection is established with the interrupter valve, the required settings for rate and depth of respiration are made,

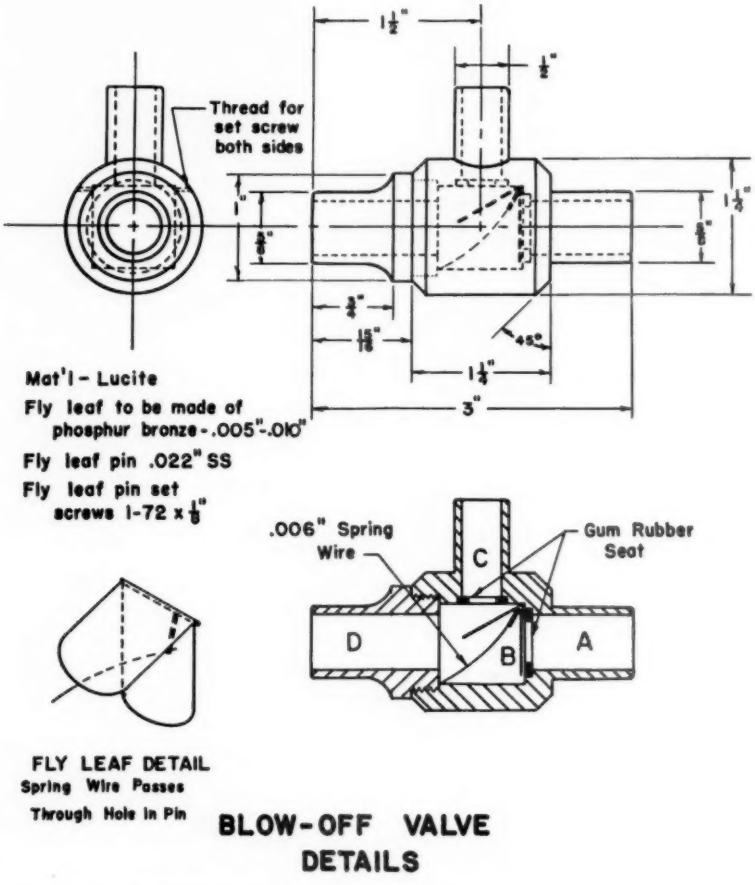


Fig. 6.—Drawing showing details of blowoff valve. When positive pressure is applied through opening at *A*, the spring-loaded butterfly valve (*B*) is forced open, and simultaneously the exhaust port at *C* is closed. This allows the anesthetic mixture to pass through *D* into the endotracheal tube. When the positive pressure is interrupted, the spring-loaded valve (*B*) resumes its resting position, closing intake port at *A* and opening the exhaust port at *C*. This permits the expired gases to escape.

and the foot pedals are set to provide a suitable mixture of ether vapor and compressed air or oxygen. If the shorter-acting barbiturate is used for induction, rapid saturation with ether is encouraged. If pentobarbital sodium is used, saturation with ether is accomplished more slowly. Shifting of the foot pedals provides any necessary change in concentration of ether vapor, depending on the depth of anesthesia required.

SUMMARY

Three modifications which have been applied to the Hunterian Laboratory anesthesia apparatus for experimental surgery have been described.

These modifications are: (1) an air reservoir which is inserted in the line between the air or oxygen supply and the ether reservoir, (2) a multiple foot pedal device for controlling the ether concentration, and (3) a blowoff valve for eliminating the expired gases.

A brief description of our anesthetic practice in experimental surgery has been presented.

Acknowledgment.—The original suggestion for the anesthesia blowoff valve described in this article was made by William H. Muller, Jr., M.D., associate professor of surgery, University of California Medical Center at Los Angeles, in whose laboratory this device was developed.

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DIAGNOSTIC PARACENTESIS IN THE ACUTE ABDOMEN

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DIAGNOSTIC paracentesis is not a new procedure. It has been used on occasion in the differentiation of the various types of peritonitis and ascites. Although there were sporadic reports of diagnostic taps in the early part of the century, the first comprehensive report was by Neuhof and Cohen in 1926.[1]

The value of diagnostic paracentesis has long been recognized in the differentiation of primary peritonitis from the peritonitis of perforated bowel, particularly in infants and children.[2] Later, its value in the diagnosis of strangulated bowel was emphasized.[3] Keith, Zollinger, and McCleery[5] stressed the importance of amylase determinations on peritoneal fluid in the diagnosis of late acute pancreatitis.

The present report stems from extensive experience over the past three years from the Tulane Surgical Service at Charity Hospital of New Orleans, La. This service includes a large group of acute abdominal cases, both traumatic and otherwise.

We have found diagnostic paracentesis to be of immense value in several groups of patients encountered. Its greatest use is in preoperative evaluation of blunt abdominal injuries, the differentiation of the various types of peritonitis, and in the diagnosis of many of the various abdominal catastrophies. The prime value, in addition to being of diagnostic import, is that it frequently averts an unnecessary laparotomy.

Blunt Abdominal Injuries.—It is generally recognized that penetrating wounds of the abdomen are an indication for laparotomy. However, the diagnostic difficulties of many of the closed injuries are well known. These injuries primarily include ruptures of the liver and spleen. The hollow viscera are not so commonly ruptured by blunt injuries, although an occasional case may be seen.

There are many patients in whom the diagnosis of severe intra-abdominal injury is obvious. These cases present no problem inasmuch as laparotomy is clearly indicated as soon as shock is combated adequately.

The patients presenting with minimal abdominal signs and symptoms frequently make the decision for or against laparotomy quite difficult. In early rupture of the spleen and liver, there is often a well-known latent period before the onset of shock. We have found that our method of paracentesis is frequently the deciding factor in these equivocal cases.

Patients are commonly seen in our emergency room with multiple severe injuries. Those with head injuries and crushing injuries of the chest are

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particularly difficult to evaluate for possible abdominal injuries. Those with head injuries are frequently unconscious or stuporous and therefore unable to cooperate for an adequate examination. We have seen patients with known head injuries who remained in shock with no physical reasons apparent. Peritoneal aspiration revealed blood which did not clot. We use this finding as a clear-cut indication for laparotomy. Ruptures of the liver and spleen are frequently seen in blunt injuries of the chest, particularly with fractures of the lower rib cage. In all of these patients, a moderate degree of upper abdominal pain and splinting is encountered. Inasmuch as these patients with severe chest injuries are poor risks from an anesthetic viewpoint, caution must be exercised in the decision for a laparotomy. Paracentesis in these cases has been invaluable in making that decision in that frequently it reveals nonclotting blood in the peritoneal cavity indicating intra-abdominal injury of a major nature.

When clotted blood is obtained from the needle, we assume that the needle has probably damaged a vessel in the abdominal wall. Blood in the general peritoneal cavity has been sufficiently defibrinated so that it will not clot.

Many patients were submitted to laparotomy with minimal or no evidence of abdominal injury other than a positive diagnostic tap. In only one patient was an unnecessary laparotomy performed. This patient was stuporous due to a head injury. Repeated needle aspiration revealed partially clotting blood from the right lower quadrant. Subsequent laparotomy revealed three needle holes in a very low right lobe of the liver extending to the iliac crest.

We know of no patient in this group over a three-year period in whom the tap was negative, who subsequently revealed major abdominal pathology.

Acute Abdominal Conditions Accompanied by Shock (Exclusive of Injuries).—A number of patients are seen here in peripheral collapse with a rigid, distended, silent abdomen, generally of recent onset. Frequently, no underlying pathology is apparent. Abdominal paracentesis has been of value in this group because it frequently distinguishes a clearly operable condition from one which is not. This pertains particularly to the differentiation of acute hemorrhagic pancreatitis from strangulated bowel.

It has been emphasized by Keith, Zollinger, and McCleery[5] that amylase levels on peritoneal fluid generally remain high after the serum amylase has fallen to normal levels in acute pancreatitis. We have had three cases in which the diagnosis of acute hemorrhagic pancreatitis was established by paracentesis with the finding of typical sanguinous, dark fluid. In these cases, all had markedly elevated amylase levels in the peritoneal fluid but had perfectly normal serum amylase levels. In these three cases, laparotomy was fortunately averted.

The hemorrhagic peritoneal fluid is likewise encountered when nonviable gut is present in the abdominal cavity. The fluids in these two abdominal emergencies are quite similar, and the amylase determination is therefore quite significant in differentiation.

The classical fluid obtained in perforated ulcer is cloudy or purulent with flecks of amorphous fibrinous material. However, the clinical picture is usually diagnostic without resort to paracentesis.

Peritonitis of Unknown Etiology.—The literature reviewed has stressed the importance of diagnostic paracentesis primarily in this group of patients. In our hands its value has been in the differentiation of so-called primary bacterial peritonitis from generalized peritonitis due to ruptured viscera in infants and children. The consensus of opinion is that smears of peritoneal exudates revealing gonococcus, pneumococcus, Staphylococcus, and Streptococcus are an indication for conservative management.

When purulent or cloudy fluid is obtained by paracentesis, a smear is indicated for the differential diagnosis. If leukocytes are present with Staphylococcus, Streptococcus, pneumococcus, or gonococcus, the diagnosis of so-called primary peritonitis is established and laparotomy is not indicated. If leukocytes alone are obtained, with or without gram-negative rods, laparotomy is performed. If numerous organisms of all types are obtained without leukocytes, it may be assumed that the needle has been in the lumen of the bowel. If the bowel is inadvertently punctured, this, per se, is not an indication for laparotomy.

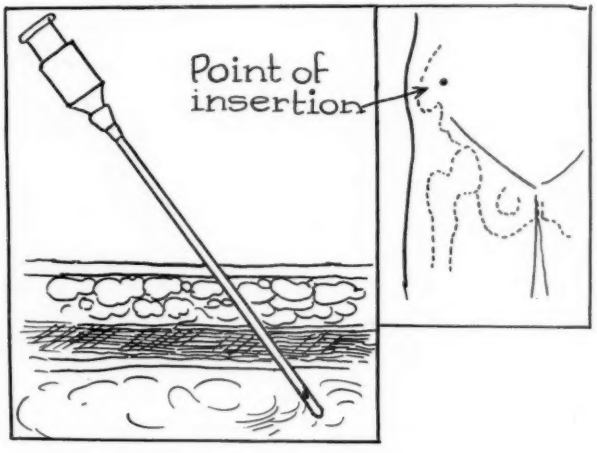

Fig. 1.—Technique.

Miscellaneous.—Clear, serous fluid has been encountered in mild pancreatitis and nonstrangulated obstructions. This type is found in ascites due to cirrhosis, heart failure, carcinomatosis, and tuberculous peritonitis, all of which are of little surgical importance. Centrifuged fluid and cell-block examinations are necessary to distinguish some of these fluids, and are out of the scope of this paper.

TECHNIQUE

The technique we use is simple. The Potter needle is probably more suitable because it is blunt and the opening is on the side, thereby not plugging as easily. However, the procedure has been carried out successfully with an ordinary 22 gauge spinal needle.

The skin is prepared in both lumbar quadrants of the abdomen and, with or without local anesthesia, the needle is inserted into the peritoneal cavity. The site of puncture is usually about 3 cm. superior and medial to the anterior superior iliac spine. The needle is directed medially into the peritoneal cavity.

The obturator of the needle is removed and, if fluid is not obtained immediately, a syringe is applied and gentle suction employed. The direction and depth of the needle may be changed without danger in an attempt to encounter fluid. The patient should be tilted so that the side of the tap is most dependent to facilitate fluid drainage. In case of a negative result, a similar procedure is used on the opposite side at the same level.

COMPLICATIONS

Needle perforations of the intestine are rare and, when they do occur, are of no consequence. In approximately 300 of these abdominal punctures, no complications have ensued. One mistaken diagnosis has been described in which an unnecessary laparotomy was performed.

CONCLUSIONS

1. Abdominal paracentesis is an important adjunct in the diagnosis of the acute abdomen.

2. In blunt injuries of the abdomen which are difficult to evaluate, paracentesis may present a concrete indication for laparotomy. This is particularly important in patients with concomitant head injuries and crushing chest injuries.

3. Patients severely ill with an acute abdominal emergency may be spared an unnecessary and even contraindicated laparotomy if the diagnosis of acute pancreatitis or primary peritonitis can be established. Evaluation of the peritoneal fluid frequently makes the diagnosis.

4. In 300 cases of diagnostic abdominal paracenteses, no complications have resulted.

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Case Reports

CANCER OF THE STOMACH: TWENTY-YEAR CURES

Report of Two Cases

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(From the Gastric Service of the Memorial Cancer Center)

THE ANECDOTE

UNTIL 1930, the therapeutic activities of the Memorial Hospital were more greatly occupied with radium and x-ray treatment of cancers. Laparotomies, except for gynecologic procedures, were seldom employed. A gastric service functioned largely as an outpatient clinic, with one or two patients seen weekly, and they were receiving only medical treatment for vague dyspepsia. In the previous decade, only three gastrectomies had been performed in the institution, and then with fatal terminations. The radical surgical treatment of cancer was frowned upon as a policy. In January, 1931, I was given the post as chief of this department, which was more nominal than actual; my status at that time and during the succeeding six months was Resident Surgeon and Fellow in Cancer Surgery. As an insurance against inexperience in technique and judgment, the one contingency on the appointment was the requirement that a senior surgeon of the hospital be in attendance as a preceptor at each abdominal operation during the first year of my incumbency.

Case 1.—The first case was of a patient with a gastric cancer, which showed no outward evidence of dissemination. At the operation, the senior consultant, who was serving as first assistant and arbiter, advised a gastroenterostomy, despite my plea for gastrectomy. His arguments were several: (a) although no hepatic metastases were seen, the cancer was unquestionably incurable; (b) gastrectomy most likely would be fatal; (c) in the presence of pyloric obstruction, gastroenterostomy would do just as well in enabling the patient to live comfortably for a few months. Accordingly, a retrocolic posterior gastroenterostomy was performed.

Twelve weeks later the senior consultant left for Europe on a vacation; with the temporary loss of surgical restriction, the patient was immediately readmitted to the hospital. A second operation was performed, consisting of a distal sleeve resection of the stomach, after the method of Billroth II. The report of the pathologist was: "Tubular adenocarcinoma. Tumor throughout thickness of wall and is growing in serosa. Not a resection case with idea of cure." Convalescence was uneventful. The patient, now 80 years of age, is living and well except for arthritis; this represents a definitive cure period of survival for twenty-two years.

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The case illustrates several points, both factual and moral: (a) The first gastrectomy for cancer performed on the newly organized gastric service of the Memorial Hospital resulted in a twenty-two-year cure. (b) A "second look" performed twelve weeks after a gastroenterostomy revealed the cancer to be still operable and permitted a second stage gastrectomy. (c) In retrospect and with regard to this one case, one might well ask which benefits the cancer patient more: the conservative attitude and greater experience of an older surgeon, or the lesser experience and more aggressive point of view of a younger surgeon? (d) The advice of Polonius to his son or the oft-quoted homily of the Golden Rule finds suitable application in this dilemma. (e) The definition of operability was established so definitively in the mind of the operator, together with the inevitable fatality without resection, that he was willing to risk censure and professional retardation if the gastrectomy had resulted in death. The subject was an indigent clinic patient. (f) The judgments of the senior surgeon with regard to operability and the pathologist with regard to curability were both incorrect, as proved by the survival of the patient for twenty-two years.

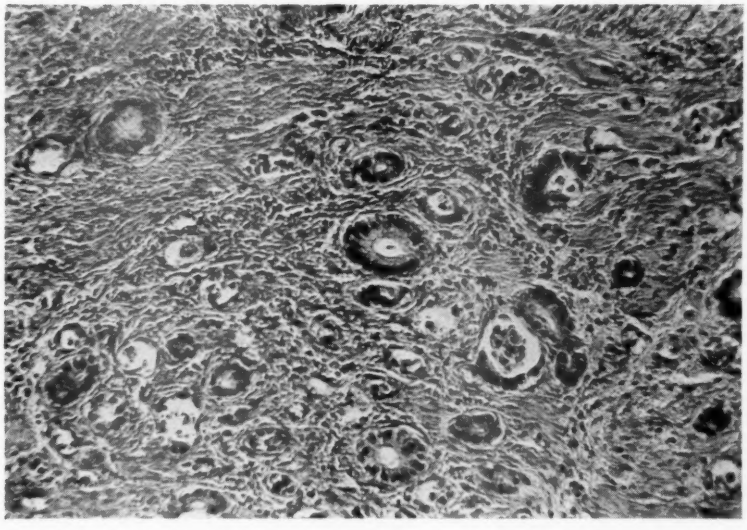

Fig. 1 (Case 1).—Tubular adenocarcinoma of stomach; twenty-two-year cure.

FORMAL CASE REPORT

J. Z., a 58-year-old man, sometime blacksmith helper and later janitor, applied to the Gastric Service of the Memorial Hospital on May 11, 1931. His past history was relatively normal except for bilateral herniorrhaphy in 1916 at the Hospital for the Ruptured and Crippled. His normal weight was 169 pounds.

Present History.—He had been in excellent health until January, 1930, at which time he developed regurgitation of bitter, hot fluid, eructation of gas, upper abdominal distress, and pain, occurring two to three hours after meals. Six hours after supper he was usually awakened with severe epigastric pain radiating from left to right, accompanied by loud gurgling. Vomiting occurred about once every two weeks. The symptoms had increased steadily since the date of onset. In December, 1930, gastrointestinal x-ray

studies made elsewhere were reported as normal. On the date of application he weighed 121 pounds with a loss of 48 pounds in sixteen months since the onset of symptoms.

Physical Examination.—The patient gave evidence of weight loss, anemia, and cachexia. No tumor mass could be felt in the abdomen. The chest was normal to physical examination. There were no palpable masses in the pelvic cul-de-sac.

Gastrointestinal x-ray series made on May 12, 1931, revealed obstruction of the pylorus with an irregular filling defect and a marked gastric retention in six hours after the barium ingestion. The diagnosis was carcinoma of the prepyloric gastric segment.

Fig. 2 (Case 2).—Roentgenogram of stomach showing early polypoid carcinoma.

Operative Procedure.—On May 29, 1931, a laparotomy was performed. The tumor was found to be confined to the distal third of the stomach. The serosa was involved, but there were no demonstrable metastases to the liver or other abdominal viscera. A retrocolic posterior gastrojejunostomy was performed. Convalescence was uneventful. After the patient was discharged from the hospital, he received some high-voltage x-ray therapy to the left epigastrium anteriorly and posteriorly, cross-firing at the distal segment of the stomach, employing 199 kv., ½ mm. of copper filter, 70 cm. target skin distance, portals 12 by 10 cm., 420 roentgens at a dose, treatment being given once every four days alternating between the anterior and posterior fields, for a total dose of 1,260 roentgens to the anterior port, and 840 roentgens to the posterior field.

The patient did not gain weight in the interval, but did have some relief of the symptoms of gastric retention due to pyloric obstruction.

He was readmitted to the hospital, and on Aug. 5, 1931, the segment of stomach distal to the previous and superiorly placed posterior gastrojejunostomy was removed, together with a liberal segment of the duodenum. This constituted a second stage Billroth II procedure. Convalescence from the second operation was uneventful.

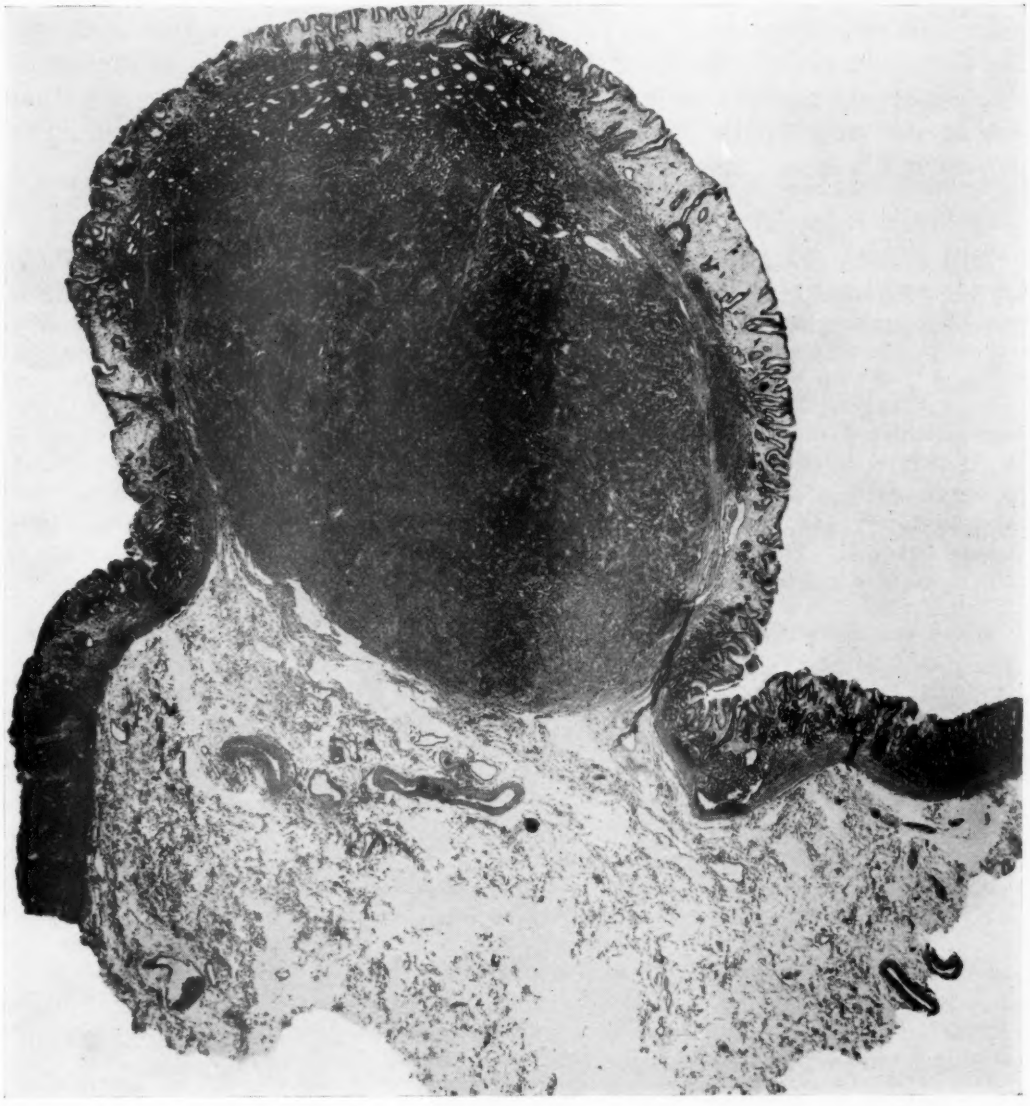

Fig. 3 (Case 2).—Low-power photomicrograph of polypoid carcinoma of stomach; twenty-year cure.

The gastric specimen was reported by the pathologist to be a tubular adenocarcinoma. In the intervening years this patient has been in good condition, except as he approached the age of 80 years he had increasing arthritis in the knees which caused him some disability. His weight increased from 121 to 144 pounds. He has had no gastric complaints, and remains in good general health for an octogenarian.

CASE 2.—Twenty to twenty-one years ago, Dr. Harry Hauser, then a Fellow of the Memorial Hospital, conducted a series of x-ray examinations

of the gastrointestinal tract in male patients over 40 years of age, who had no gastric complaints. The purpose was to ascertain, if possible, any tumors, ulcers, or other physical abnormalities which might be present in the absence of suspicious symptoms. This experiment antedated by many years the more detailed surveys recently published from numerous sources in which one patient of 800 so examined was found to have an asymptomatic gastric cancer. Dr. Hauser examined less than 100 patients and in this group discovered a single polypoid gastric tumor in one patient. Immediate surgical intervention was carried out and the tumor, an early gastric cancer, was resected. The patient is living and free of evidence of recurrence after twenty years.

FORMAL CASE REPORT

K. C. T., a 41-year-old man, was superintendent of an estate. His employer, who had endowed a bed at Memorial Hospital, sent him to the hospital for a physical examination. Since his appendectomy at age 16 years, he had taken bran, Caroid, and bile salts intermittently to regulate his bowel movements. He had a good appetite and had lost no weight. There was no nausea or vomiting. Fluoroscopy and gastrointestinal x-ray series on June 28, 1933, revealed a small circular filling defect, measuring 1.5 cm. in diameter, situated on the anterior wall of the pars media. There was no gastric residue of the barium ingesta. Gastric analysis using histamine revealed complete anacidity and no occult blood in all specimens. Laparotomy was performed on July 11, 1933, and the polypoid tumor was resected. Dr. James Ewing gave the following report: adenocarcinoma, Grade II. The patient is living and free of evidence of gastric cancer on Sept. 1, 1953, more than twenty years later.

PERFORATION OF THE STOMACH IN THE NEWBORN INFANT

A Case Report

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ALTHOUGH perforation of the stomach in the newborn infant has been a recognized entity for one-quarter of a century, only twenty-two cases have been reported. The first survival was recorded by Leger, Ricard, Leonard, and Piette in 1950.[1] Although the case herein discussed represents the twenty-third reported case and the second survival, we assume the condition is more prevalent than reported.

In the majority of the reported cases of gastric perforation in the newborn infant, the lesion is usually secondary to ulcer, mechanical injury from gavage, congestion due to asphyxia and septicemia, and, finally, congenital defects in the musculature of the gastric wall.[2] The latter was reported by Herbut[3] in 1936. Ruptures are found to occur in all portions of the stomach wall, having no one prominent location. Race does not appear to be an important factor.

Greene and Gase[4] reported the twenty-first and twenty-second cases in 1953, the first being secondary to gastric ulcer, the other to pyloric atresia. The only other report of gastric perforation secondary to pyloric atresia was made by Burnett and Halpert[5] in 1947.

CASE REPORT

C. T., a newborn Negro boy weighing 5 pounds, 8 ounces, was born at Saginaw General Hospital, Nov. 23, 1952. The infant was the product of a normal pregnancy of eight months' gestation. The mother was 23 years old, gravida iiii, para iii with negative serology. Labor lasted seven hours and resulted in a spontaneous delivery. The infant cried spontaneously and immediately.

Initial physical examination revealed a normal infant with the exception of a high-pitched cry; temperature was 98° F. rectally. He was noted to nurse poorly at first feeding. Normal meconium stools were passed on the first and second day. At 5 A.M. on the third day, dark red blood was noted in the stools, abdominal distention was first noted, and respirations became labored and shallow. Three hours later another bloody stool was passed and surgical consultation was obtained. Physical findings revealed the patient to be a dyspneic Negro male infant with increased, shallow respirations, appearing acutely ill. The lungs were normal to percussion and auscultation. The abdomen was distended and silent, but soft. On percussion, no liver dullness was detected. There were no palpable masses. Laboratory findings at this time were: white blood cells 2,700, red blood cells 7.33 million, hemoglobin 22.5 Gm., 155 per cent. Blood group was O Rh positive. Blood Kahn test was negative. Microscopic urine examination showed a rare hyalin cast and one to two white blood cells per low-powered field. At 8:45 A.M. a scout film of the abdomen in supine and upright positions revealed a large amount of air in the area of the stomach and beneath the diaphragm, suggestive of a perforated viscus.

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Intubation was carried out, without relief of the distention and without return flow of injected saline solution. While preparing for surgery, x-ray contrast studies of the upper intestinal tract were carried out, using a small amount of Lipiodol injected into the Levin tube. The Lipiodol was seen to spread diffusely throughout the abdomen through a rent on the greater curvature of the stomach.

Eight hours following onset of distention and five hours after passage of bloody stools, the infant was operated upon.

Under drop ether anesthesia, with protective warm water bottles about the body for maintenance of temperature, the infant was draped and prepared in the usual manner. Celiotomy was carried out through a right transrectus incision centered midway between the xiphoid process and umbilicus. On opening the peritoneal cavity, a large amount of bile-colored fluid was noted. A rent, about 2 cm. in length, was found along the greater curvature of the stomach and midway between the cardia and pylorus. There was no inflammatory reaction about the rent suggestive of gastric ulcer. The rent, we felt, was due to a congenital defect in the musculature of the stomach wall. The perforation was closed with a running 00 chromic catgut suture to include all layers, followed by a second seromuscular inverting layer of silk. Following closure of the stomach, the Levin tube was injected with 10 c.c. of sterile water, and no leakage occurred, confirming the presence of but one gastric defect. It was also found that the water inserted into the stomach could be expressed into the duodenum and jejunum, ruling out a congenital atresia of the duodenum. The entire gastrointestinal tract was then examined to rule out the presence of other anomalies. The wound was closed in layers, using interrupted sutures of fine silk throughout. The infant received 50 c.c. of whole blood during the procedure and was returned to the ward in good condition.

Postoperatively, the Levin suction was continued for two days, following which small amounts of 5 per cent glucose in water were given by mouth. Boiled skim milk was started on the third postoperative day with gradual increase in the formula to twenty calories per ounce. Fluid and electrolyte balance were maintained throughout, using 5 per cent glucose in water, Ringer's solution, and whole blood. Vitamin supplements of vitamin K, ascorbic acid, and Poly-vi-sol were given. Temperature remained 97° to 99° F. rectally throughout the hospital course, and the wound healed per primam. The white blood count gradually increased from 2,700 preoperatively to a peak of 27,200 on Dec. 4, 1952. Upon discharge, the white blood count was 14,700, hemoglobin 14.8 Gm., or 102 per cent. The infant was discharged on the twenty-seventh postoperative day weighing 5 pounds, 9½ ounces.

Although perforation of the stomach is uncommon, it does occur, and we consider the mortality and morbidity to be directly dependent upon early diagnosis and prompt surgical intervention. In the usual case, the infant appears entirely normal on examination at birth only later to develop feeding difficulties, melena, abdominal distention, and marked lethargy. Although the abdomen may be soft, it is distended, and the absent bowel sounds bespeak peritoneal irritation and soilage. The onset has been reported to occur from twelve hours to eleven days after birth. Diagnosis is made by the history, examination, and, most important, x-ray studies, especially upright films of the abdomen revealing free air beneath the diaphragm. We feel the use of Lipiodol advisable in contrast studies in the avoidance of complications of aspiration during and after surgery, and believe Lipiodol to offer less peritoneal reaction than barium sulfate. Injection and distention of the stomach with water after closure of the perforation avoids overlooking a second perforation and/or atresia of the duodenum, both of which have been reported.

Thorough examination of the remainder of the gastrointestinal tract is advised to rule out the presence of other anomalies.

SUMMARY

A case report of spontaneous perforation of the stomach in the newborn child, believed to represent the twenty-third occurrence of gastric perforation in the newborn infant, the second case with survival, is presented. Suggestions as to diagnosis and treatment are outlined. The mortality and morbidity depend on the rapidity of surgical intervention.

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RETROPERITONEAL RUPTURE OF THE DUODENUM DUE TO NONPENETRATING ABDOMINAL TRAUMA

Presentation of a Case and Review of the Literature

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RETROPERITONEAL rupture of the duodenum as a result of nonpenetrating abdominal trauma is an injury which occurs rarely. This injury carries a mortality of from 20 to 50 per cent although recent articles[1, 2] emphasize the fact that accurate diagnosis and early treatment will reduce the mortality almost to 0. A recent opportunity to treat such an injury successfully, coupled with the marked discrepancy between the actual and the attainable mortality in this condition, prompts this review of the subject.

CASE REPORT

K. R. A. (No. 10,898), a 29-year-old white airman, was admitted to Shaw Air Force Base Hospital, South Carolina, on April 15, 1953, following an automobile accident. He was under the influence of alcohol at the time of admission, and the history was very incomplete. Details made available during postoperative convalescence revealed that the patient was thrown against the steering wheel of his automobile at the time of the accident and that he was unconscious for a short period of time. Examination on admission was negative except for multiple contusions and abrasions about the face and knees.

I first saw the patient some ten hours after admission. At this time he complained of abdominal pain and difficulty in breathing. The abdominal pain was steady, moderately severe, and present in both lower quadrants, but most marked on the right side. The difficult breathing was attributable to right anterior chest pain, which was accentuated with chest motion. Examination was negative except for the chest, abdomen, and vital signs. The temperature was 100.4°, the pulse 124, and respirations 20. There was tenderness over the third and fourth right ribs anteriorly. Abdominal examination revealed moderate spasm of both recti in the lower quadrants, most marked on the right. There was associated tenderness without rebound in the right lower quadrant. Auscultation revealed hypoactive bowel sounds.

The white blood cell count was 23,700. Urinalysis was negative. Skull films were negative. The chest roentgenogram revealed a fracture of the right fourth rib anteriorly, and the presence of air beneath the right diaphram (Fig. 1). Scout films of the abdomen showed air retroperitoneally in the right perirenal area, sharply outlining the right kidney and having a characteristic alveolar pattern (Fig. 2). During the period of radiologic examination, the patient had two episodes of hematemesis and shortly thereafter went into shock. He was immediately given intravenous fluids and whole blood, and prepared for surgery.

Exploratory laparotomy was performed under nitrous oxide, oxygen, and ether anesthesia, with succinylcholine chloride being employed for adequate muscular relaxation. The abdomen was entered through a right paraumbilical, paramedial incision. In freeing the right rectus muscle from its fascia, a 3 by 3 cm. area of muscle contusion and softening was noted in the superior pole of the wound. A moderate amount of serosanguineous fluid was present in the abdominal cavity. Systematic examination of the abdominal viscera was negative except for these findings (Fig. 3): (1) a 10 by 10 cm. hematoma in the ascending mesocolon,

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(2) a 1 by 2 cm. area of fat necrosis on the posterior parietal peritoneum in the angle formed by the medial border of the cecum and the inferior segment of the root of the small bowel mesentery, (3) multiple serosal tears in the ascending colon, and (4) emphysema and crepitation in the retroperitoneal tissues overlying the right kidney. The entire right retroperitoneal space was exposed by incising the peritoneum on the lateral border of the ascending colon and displacing the ascending colon and its mesocolon medially (Fig. 4). The retroperitoneal tissues immediately inferior to the duodenum were bile stained. Examination of the duodenum revealed a transverse tear in the third portion of the duodenum at the point where it crosses the vertebral column (Fig. 4).

Fig. 1.—Chest roentgenogram showing air beneath the right diaphragm and fracture of the right fourth rib anteriorly.

The tear was 3 cm. in length and extended about one-half the circumference of the duodenum on its anterior-inferior surface. The tear was closed in two layers using a continuous inverting stitch of 000 catgut (Connell stitch) for the first layer and reinforcing this with a second layer of interrupted silk Lembert stitches. A Penrose drain was placed in the retroperitoneal space and brought out through a right flank stab wound. The hematoma was then evacuated from the ascending mesocolon, and the serosal tears were repaired. The colon was replaced in its anatomic position, and a layer closure of the abdominal incision was then effected. The patient received 1,000 c.c. of whole blood during the operation.

Postoperatively, the patient was treated with continuous nasogastric suction, intravenous fluids, and parenteral streptomycin and Terramycin. Oral fluids were started on the fifth postoperative day. Removal of the retroperitoneal drain was begun on the seventh postoperative day and completed on the thirteenth. On the ninth postoperative day, a partial disruption of the abdominal wound occurred. This involved the skin, subcutaneous tissues, and the anterior fascial layer; the posterior fascia and peritoneum were intact. The wound disruption was repaired under local anesthesia (bilateral intercostal nerve block with Novocain), effective closure being secured with interrupted through-and-through wire sutures.

Gastrointestinal study three and one-half weeks after operation showed a normal duodenum without evidence of constriction at the operative site (Fig. 5). The patient was discharged from the hospital at this time and returned to full military duty following two weeks of convalescent leave.

Fig. 2.—Scout film of the abdomen showing air retroperitoneally in the right perirenal area. The right kidney is sharply outlined and the air is distributed in a characteristic alveolar pattern.

INCIDENCE

Subcutaneous rupture of the intestine without penetrating injury occurs not infrequently, but duodenal involvement is rather uncommon. Siler[1] cited a review of 1,183 cases of subcutaneous rupture of the intestine in which 113 or approximately 10 per cent involved the duodenum. Maingot[3] stated that only 30 per cent of the duodenal ruptures involve the retroperitoneal portion of the duodenum. Johnson[4] reviewed the world literature from 1916 to 1944 and was able to find only fifty-two bona fide cases of retroperitoneal rupture of the duodenum. Of these patients, twenty-six survived. Also of interest is the fact that in seven of the fifty-two cases, the lesion was not discovered at

the time of surgery. Cohn[2] has tabulated the cases reported from 1944 to 1952, but some of the twenty-five cases which he listed do not involve the retroperitoneal portion of the duodenum (Siler, Case IV).

ETIOLOGY

Subcutaneous traumatic rupture of the duodenum is usually the result of blunt trauma to the abdominal wall but may also occur following blast, crushing, or run-over accidents. One of the more common mechanisms of injury is seen in automobile accidents. With rapid deceleration, the driver is forcefully thrown forward against the steering wheel, thus causing impingement of the duodenum against the vertebral column.

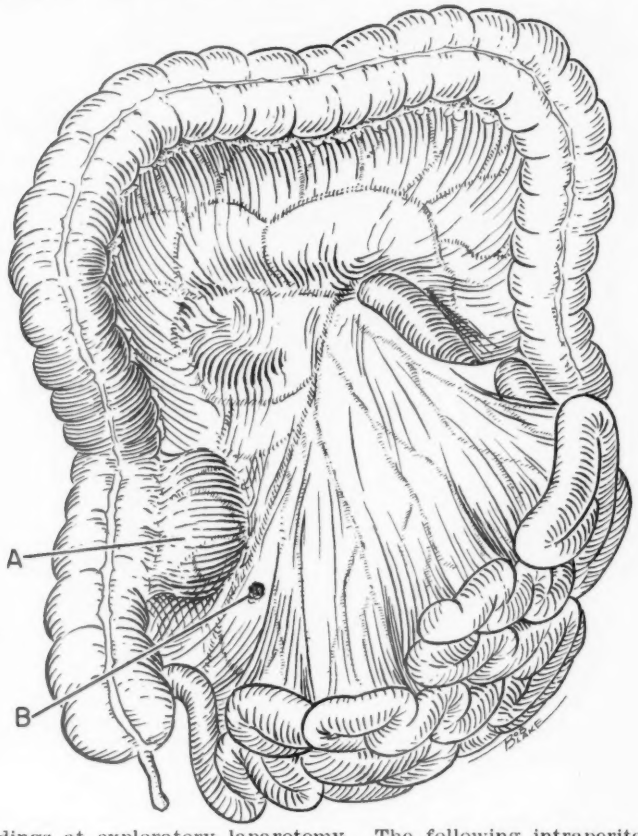


Fig. 3.—Findings at exploratory laparotomy. The following intraperitoneal lesions were indicative of retroperitoneal pathology: *A*, large hematoma in the ascending mesocolon, *B*, 1 by 2 cm. area of fat necrosis on the posterior parietal peritoneum in the angle formed by the medial border of the cecum and the inferior segment of the root of the small bowel mesentery.

MORTALITY

The case mortality rate has shown a progressive decrease over the years. Schumacher[5] in 1910 and Miller[6] in 1916 presented forty-six cases of retroperitoneal duodenal rupture with a mortality of 89 per cent. In the most recent series of cases reported,[2] the mortality rate was 20 per cent. Moreover, in this series there were no fatalities in the sixteen patients operated on between 1947 and 1952.

DIAGNOSIS

Signs and Symptoms.—When retroperitoneal rupture of the duodenum occurs as an isolated injury, the diagnosis can often be suspected from the history alone. Following abdominal injury there is a period of several hours during which the patient is asymptomatic. This asymptomatic phase is due to the fact that following the injury some time must elapse before sufficient duodenal contents spill into the retroperitoneal tissues to cause symptoms.

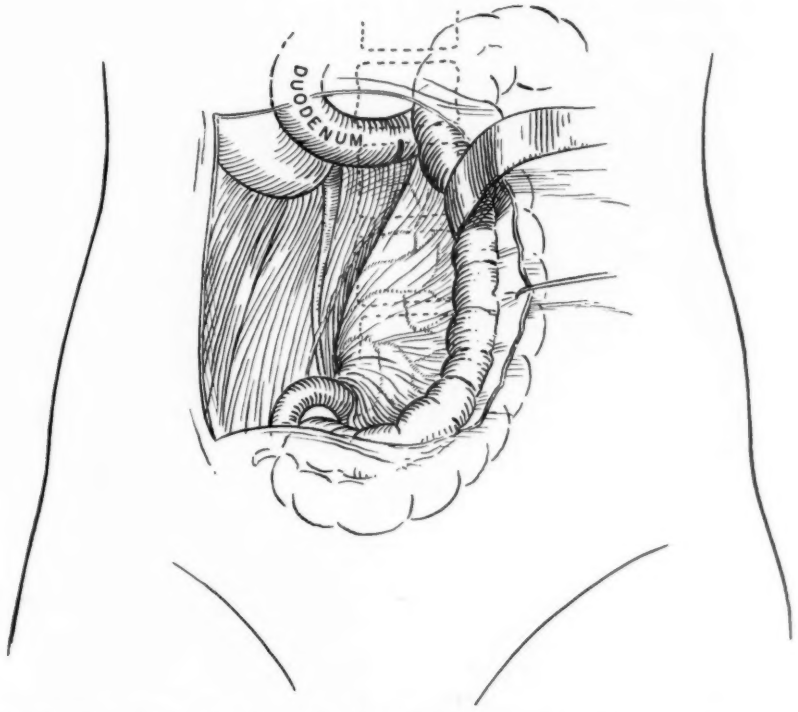


Fig. 4.—Method of exposure of the right retroperitoneal space. The peritoneum on the lateral border of the ascending colon is incised from the cecum to the hepatic flexure. The colon and its mesocolon are freed from the underlying retroperitoneal structures and displaced medially. This results in excellent exposure of the right kidney and ureter as well as the entire retroperitoneal portion of the duodenum. The duodenal tear is apparent at the point where the duodenum crosses the vertebral column.

Abdominal pain begins when sufficient duodenal leakage has occurred. This pain is steady, severe, and becomes progressively more intense. It may be localized in the right upper quadrant, in the right lumbar back area, or in the right lower quadrant.

Nausea and vomiting may or may not occur after the onset of the pain. At times, the vomitus may contain blood, although this is not a constant finding.

Irritation of the retroperitoneal tissues may include the testicular nerves, and when this occurs, testicular pain results. Testicular pain as a symptom of retroperitoneal duodenal rupture was first described by Butler and Carlson.[7] This does not appear to be a common finding, however.

Abdominal tenderness and associated muscle rigidity are usually present, and follow the course of the pain. Abdominal auscultation reveals hypoactive or completely absent bowel sounds. If the retroperitoneal escape of air from the duodenal rupture has been sufficient, emphysema and crepitation of the pelvic tissues can be detected on rectal examination.

Shock may be present and is due to the original trauma, to the leakage of duodenal contents into the retroperitoneal tissues, or to blood loss secondary to the duodenal rupture.

Laboratory Findings.—Leukocytosis is present and may vary from 10,000 to 40,000. The hemoglobin and red blood cell count are helpful in determining the amount of blood loss.

The serum amylase may be elevated as a result of traumatic pancreatitis; a moderate elevation of the serum amylase may also occur as a result of the spillage of duodenal contents retroperitoneally.

Roentgenographic Findings.—Radiologic examination of the abdomen and gastrointestinal tract is of great help in establishing a diagnosis of duodenal rupture.

Scout films of the abdomen may reveal the presence of a pneumoperitoneum. While this ordinarily suggests the rupture of an intraperitoneal hollow viscus, it may also be seen with retroperitoneal duodenal rupture. A more constant finding, as first pointed out by Sperling and Rigler,[8] is the collection of air about the right kidney and adjacent retroperitoneal tissues.

There is no unanimity of thought regarding the use of barium or Lipiodol to demonstrate duodenal rupture. Siler[1] recommended the use of barium whenever the diagnosis is in doubt.

TREATMENT

Preoperative.—In the usual case of retroperitoneal duodenal rupture, the diagnosis will not be made immediately upon admission. Radiologic studies and repeated clinical observation are required, and these take time. During this period of diagnostic work-up and observation, the patient should be at bed rest and should receive nothing by mouth. Whole blood should be available and given as indicated. Water and electrolyte balance should be maintained by parenteral administration. Continuous nasogastric suction should be instituted. Once the diagnosis has been established, operative treatment is accomplished.

Operative.—Abdominal exploration must be thorough since multiple injuries may be present. It must be remembered that there may be no intraperitoneal evidence of the retroperitoneal rupture. The following signs have been reported as suggestive of retroperitoneal duodenal rupture: (1) hematoma or bile extravasation in the ascending or transverse mesocolon, or in the retroperitoneal tissues, (2) fat necrosis of the posterior parietal peritoneum or in the ascending or transverse mesocolon, (3) crepitation or emphysema of the retroperitoneal tissue, especially in the region of the right kidney.

When any of these findings suggest duodenal exploration, the remainder of the intraperitoneal exploration should be completed first, and any lesions thus found should be treated. The duodenum should then be exposed and carefully examined. In lesions involving the second portion of the duodenum, this may easily be accomplished by dividing the posterior parietal peritoneum along the lateral border of the duodenum and then displacing the duodenum

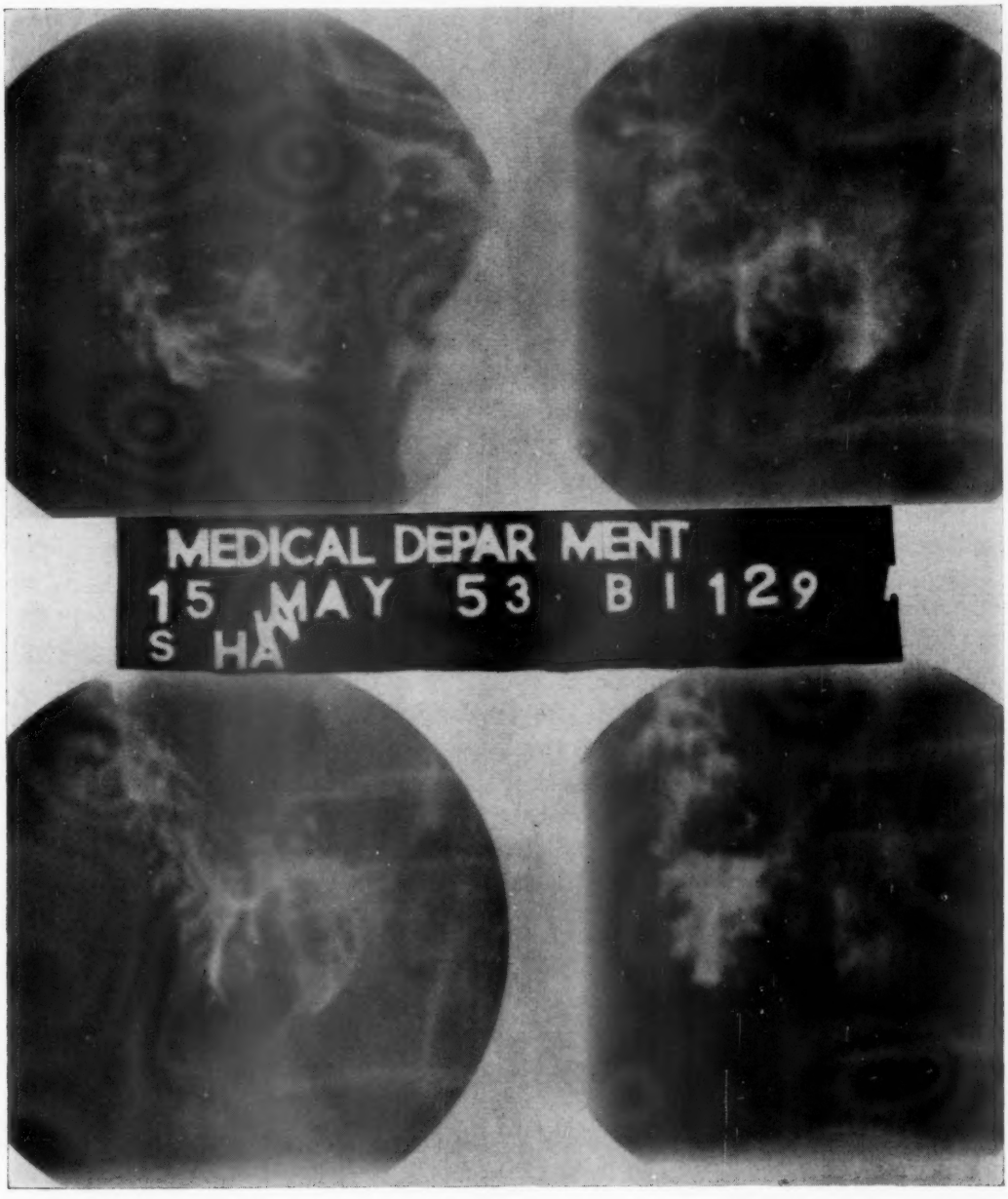
A.

Fig. 5.—Gastrointestinal x-ray study three and one-half weeks postoperatively. The site of duodenal repair is apparent, but there is no evidence of constriction or obstruction at the operative site.

to the left. In those cases involving the third and fourth portions of the duodenum, and in those cases in which the exact nature and extent of the retroperitoneal injury is not immediately apparent, greater exposure seems desirable. Complete exposure of the right retroperitoneal space, including the entire retroperitoneal portion of the duodenum, is easily obtained by incision of the peritoneum on the lateral border of the ascending colon and displacement of the colon and its mesocolon medially (Fig. 4).

The local treatment of the duodenum will depend on the nature and extent of the perforation. Whenever feasible, simple suture of the perforation

should be accomplished. Such closure is best obtained with a Connell type inner layer closure with catgut, reinforced with an outer layer of nonabsorbable Lembert or Halsted sutures. When such closure would result in stricture



B.

Fig. 5*B*. (*For legend see opposite page.*)

at the site of repair, the choice of treatment lies between the following: (1) complete division of the duodenum with end-to-end anastomosis, (2) complete division of the duodenum, closure of both ends, and gastrojejunostomy, (3) division of the duodenum, closure of the distal duodenal stump, and duodenojejunostomy.

Drainage of the retroperitoneal space is mandatory and is best accomplished through a stab wound in the right flank.

Postoperative.—Treatment here is similar to that following any major gastrointestinal surgery. Water and electrolyte balance are maintained by parenteral administration.

Continuous nasogastric suction is employed until the return of normal peristalsis is apparent. Parenteral streptomycin and Terramycin are used for such time as is indicated by the clinical course.

COMPLICATIONS

The most commonly encountered complications following retroperitoneal duodenal rupture are peritonitis and retroperitoneal cellulitis. Other complications include duodenal fistula, pancreatic fistula, subphrenic and subhepatic abscess.

SUMMARY

1. Retroperitoneal rupture of the duodenum due to nonpenetrating abdominal trauma is an injury which occurs rarely. The mortality rate is high unless accurate diagnosis and early surgical treatment are accomplished.

2. Diagnosis is based on an accurate history, repeated clinical observation, and radiologic findings. There are no constantly present pathognomonic signs or symptoms in this type of injury

3. At laparotomy, there may be no intraperitoneal evidence to indicate the retroperitoneal pathology. However, there are findings which when present are indicative of retroperitoneal pathology.

4. The method of treatment of the duodenal rupture is dictated by the extent of the injury. Intestinal continuity should be re-established by the simplest procedure possible. In the majority of cases, this can be accomplished by transverse closure of the duodenal tear.

5. Drainage of the retroperitoneal space is mandatory.

6. Postoperative treatment is similar to that employed following any major gastrointestinal surgery.

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TOTAL ESOPHAGOPLASTY USING INTRATHORACIC RIGHT COLON

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THE problem of reconstruction of the esophagus following resection has remained a major difficulty since Torek[1] in 1913 reported the first successful case of resection of the thoracic esophagus. This paper reports the intrathoracic transplantation of the right and transverse colon following total thoracic esophagectomy in one patient with carcinoma of the esophagus.

The use of stomach or tubes formed from stomach has been one of the most common methods of reconstruction of the esophagus. The stomach has been transplanted subcutaneously by Jianu,[2] Fink,[3] Kirschner,[4] Beck,[5] Halpern,[6] Hirsch,[7] Kümmell,[8] and others. Modifications of this method have been reported more recently by Mes,[9] Rienhoff,[10] and Potts.[11] The first intrathoracic reconstruction using stomach following a resection of a carcinoma of the lower third of the esophagus was reported by Adams and Phemister[12] in 1938. Since then, higher and higher transplantation has been accomplished, culminating in the work of Garlock,[13] Sweet,[14] and others who have performed a gastropharyngeal anastomosis.

Antethoracic jejunal transplantation was suggested as early as 1904 by Wullstein[15] and was successfully used by Roux[16] in France and Herzen[17] in Russia during the year 1907. There have been numerous modifications of this method by many surgeons since that time either with or without a short skin tube connecting the cervical esophagus with the proximal end of the transplant. Intrathoracic jejunal transplantation has been a more recent accomplishment as reported by Rienhoff,[18] Robertson,[19] Harrison,[20] and others. The use of a subcutaneous skin tube to bridge the cervical esophagus and stomach was first proposed and utilized by Bircher in 1894.[21] This has been used intermittently to the present day. One modification was that of Esser,[22] who in 1917 described two cases in which inlay Thiersch grafts were placed on a stent in a subcutaneous tunnel. The use of intrathoracic skin tubes was accomplished by Bricker and associates[23] in 1949.

Pathologic study of carcinoma of the esophagus has demonstrated repeatedly that segmental resection is inadequate and that recurrence due to intramural spread up and down the esophagus occurs with disturbing frequency. In a recent report[24] of the experience at Memorial Center and New York Hospital, 45 per cent of a group of seventy-nine esophagectomies were inadequately resected as determined by histologic evidence of spread up and down the

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esophagus, an additional 35 per cent were inconclusive as to adequacy of resection, and in only 20 per cent was there definitely an adequate resection. Ochsner[25] collected 1,025 autopsied cases of carcinoma of the esophagus and stated that in 41 per cent the lesion had not extended outside the esophagus. Abel[26] reviewed another series of fatal cases of carcinoma of the esophagus and found that 25 per cent of these were operable. These observations seem to indicate that it is not futile to hope for some improvement in results following more extensive resections. All the published results using present methods of treatment (including radiation) show that the prognosis is so poor that there is an obvious need for improvement. Any procedure in which a very wide margin of esophagus is not taken is to be condemned. The fear of inability to reconstruct the esophagus often causes a surgeon to perform an inadequate resection. It would be of real value to have available for reconstruction some structure of adequate length with good blood supply to replace the entire esophagus. We believe that colon might serve such a purpose. It would then be possible to extend the limits of resection further by excision of parts of the trachea, or great vessels, and by radical neck dissection. Should the use of colon as a transplant stand the test of time, it should prove especially valuable for lesions of the upper esophagus, whether benign or malignant.

The chief advantage of stomach is that it is readily available. However, it is very difficult to bring it well up into the neck and there is the constant temptation to make the proximal margin of resection too close to the tumor. Furthermore, the esophagus does not tolerate gastric secretions well, and peptic esophagitis of a very serious degree may develop. The vagotomized stomach may undergo dilatation and become atonic, causing encroachment on the surrounding tissues which may embarrass pulmonary function. This is especially true in infants, where it is used for correction of atresia. These patients may regurgitate gastric juice into the pharynx and aspirate, particularly with cervical anastomosis. There have been successful cervical gastric anastomoses, but they are the exception rather than the rule.

We have had little direct experience with skin tubes, and although at first thought they would seem to be of considerable value, in reality the disadvantages are numerous. They usually require a great many stages and long hospitalizations, with numerous small failures and disappointments. Strictures and fistulas at either end are common, and at the gastric end these are particularly difficult to treat.

Because of the inadequacies of both stomach and skin tubes, the use of jejunal loops has been extended, and ingenious methods have been devised to make their use successful. The great disadvantage of using jejunum is that it is extremely difficult and often impossible to preserve a blood supply of sufficient length to nourish the transplant all the way to the neck. Despite extensive investigation to offset this disadvantage, almost everyone who has had much experience with esophagoplasty admits that the blood supply is still a major problem. Mes[9] in his review stated that more jejunal transplants perish than survive. Yudin,[27] who has probably had more experience by far than

anyone else (having performed some 200 to 300 esophagojejunostomies by 1949[28]), stated, "Total intestinal plastics can be very difficult, very risky, or absolutely impossible" and "There are some cases in which the greatest skill is doomed to failure; the arcades are too short, their junctions are extremely thin, the radial trunks are in many places insufficiently developed, unequally distributed, the mesentery is too fat and easily tears." Shumacker[29] stated that the reported incidence of failure due to poor blood supply is quite high, but the real incidence is probably much higher. Other difficulties include ulcerations from intestinal secretions. If jejunum is anastomosed into the stomach distally, the incidence of jejunal ulceration is quite high; so high, in fact, that most surgeons by-pass the stomach, using a Roux Y transplant. If no food is going into the stomach, the isolated gastric pouch seems to be an invitation to peptic ulceration.[30] Yudin[27] does not feel that by-passing the stomach leads to any serious consequences, although he does not make any definite statement regarding peptic ulceration but refers rather to "metabolic disturbances," and so forth. Sweet[31] has objected to the Roux Y procedure, stating it may have all the disadvantages of a jejunostomy.

Inert tubes (rubber, plastic, and so forth) as stents to bridge the gap following esophageal resection have been used infrequently since 1922.[32, 33] Epithelization occurs around the tube, which is later passed or removed from above, leaving a patent tube. These have been essentially palliative procedures used to bridge short gaps and have not worked out as well as was hoped at first.

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The use of colon in this country has been practically negligible. Last fall, Scanlon, Morton, and co-workers[47] at Memorial Center attempted the procedure in two patients who died in the immediate postoperative period, although the bowel was viable at the time of death in both. Early in 1953 a surgeon at Children's Hospital at Los Angeles is said to have used transverse colon in the reconstruction of an infant with esophageal atresia. The results of this procedure are not available to us at the present time. Last year Wangensteen,[48] in discussing Ravitch's paper, stated that in patients under 50 years of age with an active gastric secretion, colon is probably a far better organ to use than

esophagus, an additional 35 per cent were inconclusive as to adequacy of resection, and in only 20 per cent was there definitely an adequate resection. Ochsner[25] collected 1,025 autopsied cases of carcinoma of the esophagus and stated that in 41 per cent the lesion had not extended outside the esophagus. Abel[26] reviewed another series of fatal cases of carcinoma of the esophagus and found that 25 per cent of these were operable. These observations seem to indicate that it is not futile to hope for some improvement in results following more extensive resections. All the published results using present methods of treatment (including radiation) show that the prognosis is so poor that there is an obvious need for improvement. Any procedure in which a very wide margin of esophagus is not taken is to be condemned. The fear of inability to reconstruct the esophagus often causes a surgeon to perform an inadequate resection. It would be of real value to have available for reconstruction some structure of adequate length with good blood supply to replace the entire esophagus. We believe that colon might serve such a purpose. It would then be possible to extend the limits of resection further by excision of parts of the trachea, or great vessels, and by radical neck dissection. Should the use of colon as a transplant stand the test of time, it should prove especially valuable for lesions of the upper esophagus, whether benign or malignant.

The chief advantage of stomach is that it is readily available. However, it is very difficult to bring it well up into the neck and there is the constant temptation to make the proximal margin of resection too close to the tumor. Furthermore, the esophagus does not tolerate gastric secretions well, and peptic esophagitis of a very serious degree may develop. The vagotomized stomach may undergo dilatation and become atonic, causing encroachment on the surrounding tissues which may embarrass pulmonary function. This is especially true in infants, where it is used for correction of atresia. These patients may regurgitate gastric juice into the pharynx and aspirate, particularly with cervical anastomosis. There have been successful cervical gastric anastomoses, but they are the exception rather than the rule.

We have had little direct experience with skin tubes, and although at first thought they would seem to be of considerable value, in reality the disadvantages are numerous. They usually require a great many stages and long hospitalizations, with numerous small failures and disappointments. Strictures and fistulas at either end are common, and at the gastric end these are particularly difficult to treat.

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stomach, although the technical difficulties in employing it rather than stomach are obvious. Ochsner,[49] in a review of all types of esophagoplasties, stated that of all the groups in which the operation was started and the end result stated, colon was best, with 56 per cent good results. The colon group showed the lowest mortality rate, namely, 22 per cent. Ochsner further stated that the advantage of using colon in reconstruction is the ease with which a relatively long loop, the viability of which is definite, can be secured.

The crucial factors in utilizing colon are: (1) Is the anatomy and blood supply adequate to produce a sufficiently long segment without question of viability and (2) will the colon resist gastric secretions? Both right and left colon transplants have been accomplished, and the blood supply to each has been studied. The marginal artery to the colon is fairly close to the bowel wall, as contrasted with small bowel. In about 5 per cent of cases, this artery is incomplete near the ileocecal junction, but there are usually anastomoses nearer the root of the mesentery to offset this. In a series of autopsies, Toupet[41] found that the middle colic artery will supply adequate blood to the left colon in all cases to reach the level of the pharynx. Fry[59] dissected twenty human cadavers and found adequate length of blood supply to the right colon in all. Scanlon and Morton[47] dissected sixteen cadavers and found that the left colon would always reach the pharynx. When right colon was evaluated, in four of the sixteen cases it would not reach above the clavicle, but there were unusual anatomic features present in these four cases. The right colon could not be mobilized adequately because of a large liver in three, and the fourth was a six-foot, two-inch man whose cecum lay high in the right gutter. We autopsied two infants and three adults and found that the cecum would reach to the pharynx in all after division of the ileocolic vessels. We have carried out no experimental work of our own to demonstrate resistance of colon to gastric secretion, but Dragstedt and Vaughan[51] in 1924 took a loop of dog's colon and sewed it into the stomach as a flap. After one year the colon was normal both macroscopically and microscopically. Ivy,[52] Wangensteen,[53] and Florey and Harding[54, 55] have all shown the resistance of colon to gastric secretion to be greater than that of jejunum or esophagus, and Moroney[56] has transplanted colon into stomach in twenty-two ulcer patients to act as a reservoir.

CASE REPORT

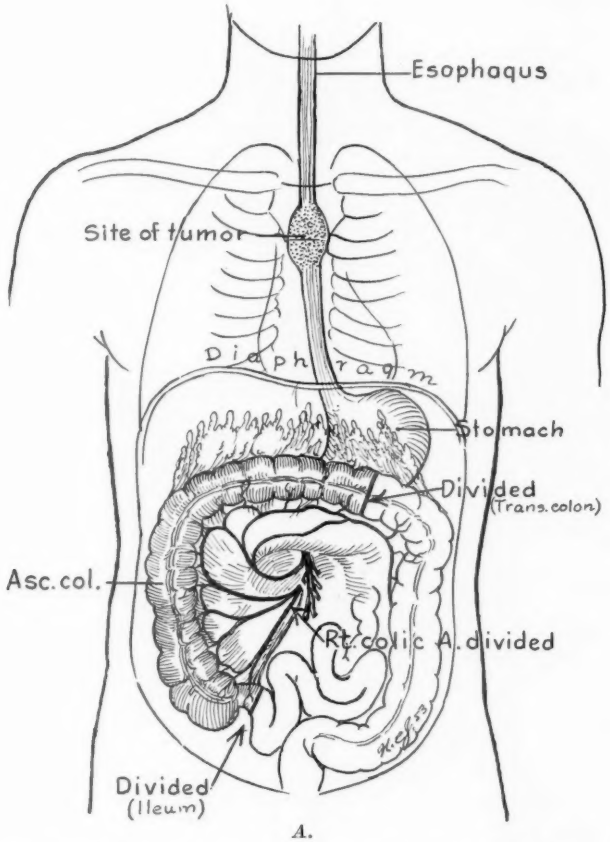
W. C. (SMH No. 355947), a 55-year-old white man, was admitted to Strong Memorial Hospital on Jan. 15, 1953, because of difficulty in swallowing. In September, 1952, he had first noted this symptom and had consulted his physician. A barium swallow was performed, and no abnormality of the esophagus was noted. Dysphagia gradually increased and he was admitted for esophagoscopy.* He had lost twenty pounds in weight but had no other symptoms. The past history and family history were noncontributory.

Physical examination was essentially negative except for evidence of weight loss. Blood pressure was 140/86. Heart and lungs were negative. Laboratory findings were: hemoglobin, 15 Gm.; hematocrit, 44 per cent; white blood count, 8,800 with normal differential; Wassermann, negative; total proteins, 6.5 Gm. per cent with normal albumin-globulin ratio; nonprotein nitrogen, 25 mg., alkaline phosphatase, 10.9; prothrombin activity, 80 per

*By Dr. C. Heatly.

cent; Na, 140 meq.; K, 4.65 meq., Cl, 108 meq., electrocardiogram normal. Barium swallow showed a filling defect in the esophagus from the level of the third to the sixth dorsal vertebrae. Esophagoscopy showed an irregular mass involving the esophageal wall at the beginning of the thoracic esophagus. Biopsy showed epidermoid carcinoma.

The preoperative preparation included large bowel preparation with Sulfathalidine and neomycin.



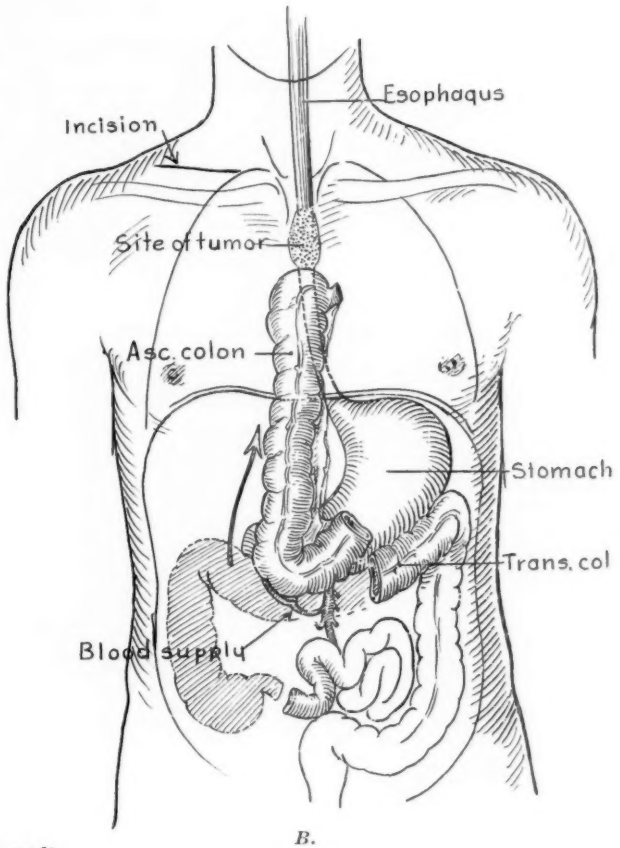
A.

Fig. 1.—*A*, Note that the marginal artery is quite close to bowel so that its length is almost equal to the length of colon it supplies. The ileocolic artery is divided near its origin. Note the point of division of ileum and transverse colon.

Operation.—Operation was performed Jan. 27, 1953. (See Fig. 1, *A*, *B*, and *C*.) Anesthesia was Pentothal Sodium and I.T. gas-oxygen-ether. The right pleural cavity was entered through the bed of the sixth rib. The carcinoma of the esophagus extended from the level of the first rib posteriorly to below the azygos vein. It was adherent to the membranous portion of the trachea. The entire esophagus was mobilized and the thoracic wound temporarily closed. The patient was turned on his back, and the abdomen was entered through a midline incision. The cecum, ascending, and transverse colons were then mobilized. After digital compression of the right ileocolic artery it was divided at its origin from the superior mesentery artery. The terminal ileum was divided and the distal end inverted. The transverse colon was divided and the continuity of the bowel restored by end-to-end ileocolostomy. The free segment of bowel maintained normal color, with the middle colic artery as its only source of blood supply. The distal end of the segment (transverse colon) was then anastomosed to the anterior wall of the stomach.

The thoracotomy wound was then reopened, the esophagus divided at the cardia, and distal end inverted with two layers of silk. The esophagus was then brought out through

an incision in the neck. A tunnel was then made through the anterior mediastinum by blunt and sharp dissection, and the cecal end of the free colon segment was brought up and anastomosed to the divided esophagus in the neck. The anastomosis between the esophagus and cecum was performed with two layers of fine silk. The colon was under no tension and the color of the bowel was excellent. The wounds were closed and both pleural cavities were drained with intercostal catheters attached to water-bottle suction because of bilateral pneumothorax.



B.

Fig. 1 (*Continued*).

B, The appendix has been excised and the terminal ileum turned in. The colon is being brought to the neck with ease through the retrosternal tunnel. Not shown on the drawing is a branch of the middle colic artery which supplied the distal segment of transverse colon.

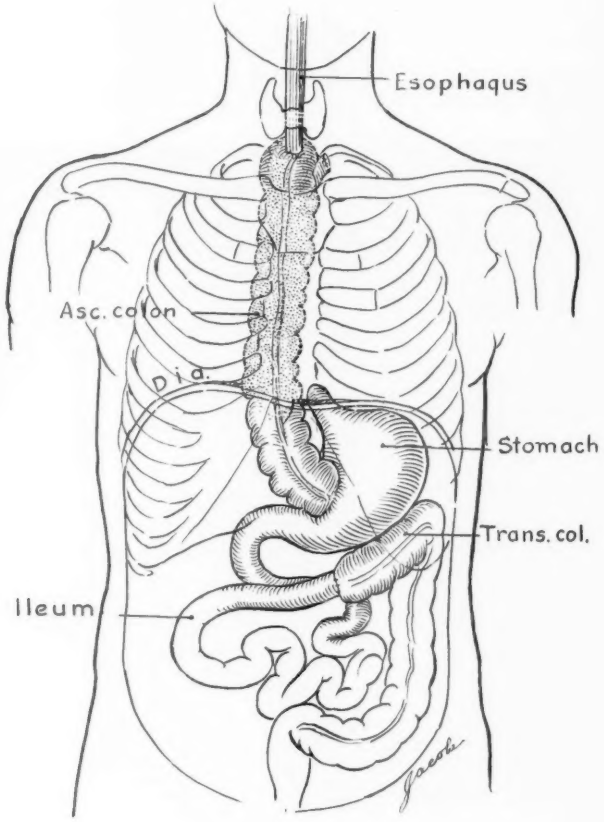
The postoperative course was stormy for two days but then improved, and by the eighth postoperative day, the patient was tolerating full liquids and soft solids by mouth with no difficulty in swallowing. On the twelfth postoperative day, low-grade fever developed and an abscess of the lower pole of the cervical incision was drained. This was due to a fistula with the esophagus. The fistula closed spontaneously within six days and the patient was again able to take full liquids. He was discharged one month after the operation.

On March 10, 1953, the patient was readmitted because of difficulty in swallowing. (See barium swallow, Fig. 2.) He had been able to take liquids and solids following discharge, but for two days had been having increasing difficulty in swallowing, and was losing weight. A feeding gastrostomy was contemplated but the colon transplant overlay the stomach; so a feeding colostomy was performed. Following this, the patient gained weight and was able to swallow liquids without difficulty, but continued having difficulty

with solid food. During April he received sectional rotational x-ray therapy with the million-volt machine.

On May 14, 1953, biopsy of a subcutaneous nodule below the right scapula showed epidermoid carcinoma. The liver was enlarged at this time.

On May 20, 1953, the patient suddenly developed pain in the chest with marked dyspnea, and he expired within twenty-four hours. It was felt that the trachea had been perforated, but no autopsy could be obtained.



C.

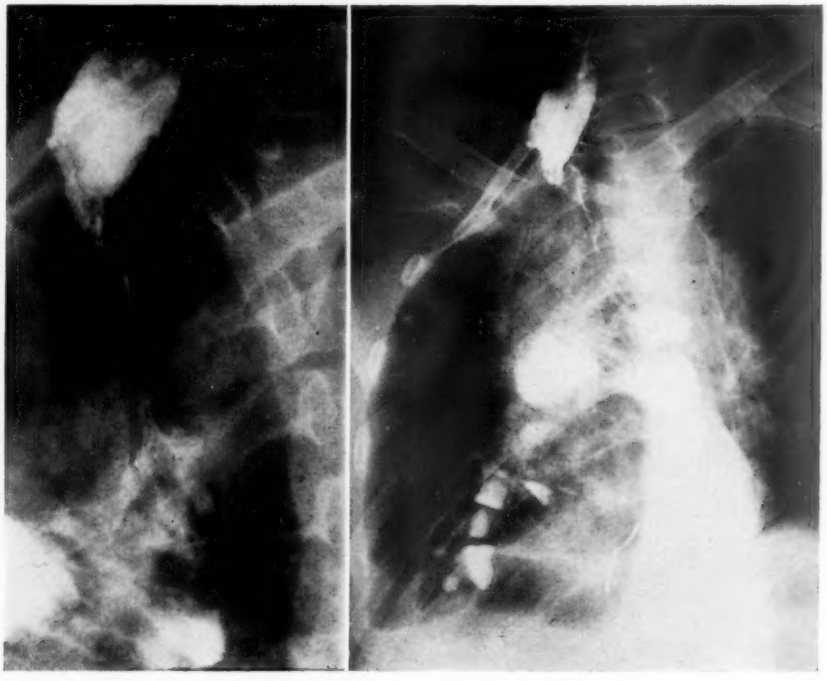
Fig. 1 (*Continued*).

C, The completed operation. The entire thoracic esophagus has been excised. The cervical esophagus has been anastomosed to cecum. The proximal transverse colon has been anastomosed into the anterior wall of the stomach and an end-to-end ileotransverse colostomy has been completed. The sole blood supply to the transplanted colon comes from the middle colic artery.

DISCUSSION

This one case report supports the feasibility of using colon to replace the esophagus. In this instance the lesion had involved the trachea, and it recurred rather promptly. The patient did, however, have a postoperative period of freedom from symptoms, and during this time he was surprisingly free of digestive symptoms. In our experience, the patients usually have loss of appetite, nausea, and other distressing symptoms when the stomach is transplanted high in the chest. These symptoms may be avoided if the stomach is left in its normal position, as in this procedure.

The operative procedure involves both a thoracic and abdominal incision, but with further experience in the technical aspects, the procedure should be less formidable. The thoracic portion can be completed first, and if the patient's condition is poor, the colon replacement can be delayed as a second stage. The procedure should be especially valuable for lesions in the middle and upper thirds of the esophagus. The technical procedure is feasible, it should carry a low mortality, and the physiologic functional aspects appear superior to other procedures. The greater length of the colon which is available for transplantation should make possible more adequate resection of carcinoma of the esophagus.



A. *B.*

Fig. 2, *A* and *B*.—Barium swallow seven weeks postoperatively. There is a partial but not complete stricture just above the clavicle at the esophagocecal anastomosis.

SUMMARY

The utilization of a retrosternal colon transplant for reconstruction of the thoracic esophagus following esophagectomy for carcinoma is reported. A brief historical review of esophagoplasty and the rationale of esophageal reconstruction with colon are presented. We feel this method has theoretical and practical advantages over other types of esophagoplasty now in use, especially for carcinoma of the upper thoracic esophagus. Further trial seems warranted.

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SIMULTANEOUS ACUTE OBSTRUCTION OF THE COLON AND TERMINAL ILEUM FROM ENDOMETRIOSIS

RICHARD JOSEPH WHITE, M.D., FORT WORTH, TEXAS

ENDOMETRIOSIS is a very common disease, and the familiar picture of relative sterility, acquired dysmenorrhea, deep pelvic pain, palpable tender nodules in the vaginal fornix or cul-de-sac, frequent tender ovarian masses, and dyspareunia is known to every surgeon who is concerned with gynecology. Many of us have seen the blue or puckered spots on the lower small intestine, sigmoid, and rectum along with nodules in the cul-de-sac, hemorrhagic cysts of the ovaries, and, at times, woody adhesions of the rectum to the posterior cervix which give pause to the most ardent advocates of the routine use of complete hysterectomy, but the picture of intestinal obstruction from endometriosis is not so generally known. McGuff, Dockerty, and others[1] assembled only sixteen cases from the files of the Mayo Clinic and Colcock and Lamphier,[2] fourteen from the Lahey Clinic. A few small series have been gathered here and there, and there are a number of single case reports, but, as Dockerty pointed out, a "literature" of only forty cases at the time of their report emphasizes its rarity or its lack of recognition. The preponderance of these cases has been in the sigmoid or rectosigmoid, with a few in the terminal ileum. The rectum is often involved but, because of its large size, is rarely obstructed. I have found no account of acute obstruction of the terminal ileum and the sigmoid at the same time from endometriosis, and wish to report such a case.

M. B., a 44-year-old single woman, was admitted to St. Joseph's Hospital March 13, 1950. (Discharge was on May 28, 1950.)

Some eight days before admission here, this patient had been seen in a hospital in a neighboring town as an emergency with a history of cramping and vomiting and no bowel movement for several days and a gradually developing abdominal distention. Plain plate of the abdomen showed marked distention of the colon and small bowel. There had been no passage of blood or mucus by rectum, and nothing could be felt in the rectum. There was a vague sense of thickening in the vaginal vault, but because of the great distention, the uterus could not be felt. She gave a history of many attacks of abdominal cramps, severe constipation, and sometimes abdominal distention at menstrual time during the previous several years.

Endometriosis was discussed as a possibility, but I thought carcinoma of the left colon more likely. She was operated on as an emergency through a right mid-rectus incision; all the small bowel and the colon were hugely distended. A hard mass in the rectosigmoid which felt like carcinoma was palpated. A loop of transverse colon was freed and brought out over a glass rod, the wound was closed about it, a purse-string suture was placed, and through a large needle a great quantity of gas was evacuated. The next day her physician opened the colostomy with a cautery. Several days passed, and there was very little passage of gas and feces from the colostomy, and the cramps, nausea, and distention persisted, with partial relief by suction. I saw the patient again after one week, when she was transferred to St. Joseph's Hospital by ambulance. A barium enema was

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performed on April 14, 1950, and examination of the colon by retrograde enema revealed a complete obstruction to the flow of barium at about 17 cm. from the anus. Re-examination, on April 19, 1950, by injecting barium into the distal colostomy loop showed that the mucosa was normal to the area of the previously described obstructing mass. Here, loops were rather redundant, but actual malignancy could not be identified. Bowel filled distal to this and the rectum was normal. From the general appearance, it was felt that this was inflammatory; however, perforated carcinoma could not be ruled out.

A day or two later there was profuse drainage of thin fecal contents through the colostomy with marked recession of symptoms, but this was short lived. Gradually increasing signs of complete small bowel obstruction developed, and re-exploration was carried out on April 19, five days after admission, through a long left rectus incision because of infection in the original incision. Enormous loops of dilated small bowel were encountered. A purse-string suture was placed, and through a stab wound a trocar was inserted; by suction and pleating of the bowel, a large quantity of gas and bowel content was evacuated. The bowel was traced down practically to the ileocecal junction. Dense fibrous scar was encountered in the pelvis which did not feel like malignant tissue, and since it seemed certain that an attempt at dissecting loose the last three or four inches of the terminal ileum involved in this mass would result in a perforation of the bowel and a massive spill, a lateral anastomosis to the now collapsed cecum was performed with two layers of Dulox sutures. The mass in the rectosigmoid was re-examined, and it felt like a carcinoma. No gross evidence of metastatic spread was seen. The patient's condition was poor at the end of this procedure, but she rallied and soon began to have profuse bowel movements through the colostomy. After she was convalescent, a proctoscopic examination was made and a rather tight stricture in the lower sigmoid was encountered which could not be passed. The mucosa seemed intact, and no gross evidence of carcinoma could be seen. She was allowed to recuperate from the almost fatal double obstruction until May 5, 1950, and after proper preparation with sulfasuxidine and bowel cleansing, she was operated on for the obstructing tumor of the rectosigmoid. After separation of inflammatory adhesions, massive woody-feeling plaques were found in the pelvis, so that it seemed impossible to make a safe dissection to clear the pelvic floor for consideration of an abdominoperineal resection. The tumor of the rectosigmoid felt very hard and was constricting, but extending from it and continuous with it for about one and one-half inches outside the bowel down over the right sacrum was a woody, hard, stringlike mass. Since it seemed impossible to consider a wide resection if the lesion were malignant, it was decided to try to remove the obstructing lesion and perform an end-to-end anastomosis, preserving the superior hemorrhoidal vessels, if possible. The mass was resected very close to the bowel wall. There was fair bleeding from both bowel ends. End-to-end anastomosis was performed, and she had an uneventful recovery.

*Pathologic Report.**—Gross sectioning of a segment of large bowel some 11 cm. in length revealed the mucosa to be intact. Deep within the mid-part of the wall was felt a firm, elastic tumor mass, and gross sectioning of this revealed an unencapsulated grayish white tumor with radially arranged strands. Deep within the mass was a yellowish white mottled area within the fatty mesentery, within which short yellowish white masses were found. Sections showed dense fibrous tissue as forming the major portion of the blocks. This was practically hyaline in areas, with the strands cut in all directions and cut at various angles. Bits of intestinal muscularis were recognized. Deep within were found gland tubules lined with low to tall cells resembling those from endometrium. Elsewhere and within the yellow portion mentioned previously was seen marked fibroplastic tissue infiltration, with numbers of lymphocytes and a smaller number of leukocytes. There was much congestion here.

Diagnosis was endometrioma of bowel.

*By Dr. Sim Hulsey.

Three weeks later the transverse colostomy was closed. When the patient had recovered from this, she was given a sterilizing dose of x-ray. She made a rapid recovery, and except for two minor episodes of abdominal cramping has been well since.

SUMMARY

1. The rarity of bowel obstruction from endometriosis is discussed.

2. A case, believed to be unique, of simultaneous acute obstruction of the terminal ileum and rectosigmoid from endometriosis is presented.

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NECROTIZING PANARTERITIS FOLLOWING REPAIR OF COARCTATION OF AORTA

Report of Two Cases

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THE occurrence of periarteritis nodosa is generally considered a phenomenon related to hypersensitivity.[1] Lesions resembling those of periarteritis nodosa in man have been produced in experimental animals by many methods, including those involving chiefly increased arterial blood pressure.[2] That hypertension may play an important role in human periarteritis nodosa has been suggested.[3] Two clinical cases are reported here in which lesions of necrotizing panarteritis indistinguishable from periarteritis nodosa developed following the surgical repair of a coarctation of the aorta. One of these cases has come to autopsy. The distribution of the lesions in vessels subjected to suddenly increased pressure has suggested the possibility that vascular strain is important in their production.

CASE REPORTS

Case 1.—A white boy, 12 years of age, was first seen at the University Hospitals on June 4, 1953, with complaints of prominent pulsations in the neck and aching in the legs following exercise. There were histories of asthma, hypertension, and rheumatic fever in his family. The patient had had no serious illness or evidence of hypersensitivity.

Physical examination showed prominent pulsations in the lower anterior neck to the right of the midline. There was a blowing systolic murmur over the precordium. The blood pressure in the arms was elevated to 160/98. Femoral pulses were barely palpable. Fluoroscopy showed left ventricular enlargement and bilateral rib notching. The leukocyte count showed 6,000 cells, of which 70 per cent were neutrophils, 23 per cent lymphocytes, 3 per cent monocytes, 2 per cent eosinophils, and 2 per cent basophils. The hemoglobin was 14.0 Gm. per 100 c.c. Urinalysis was negative; body temperature was normal.

On June 26, 1953, under Pentothal Sodium anesthesia, a coarctation of the aorta was excised and an end-to-end anastomosis made. The operation was uneventful and required three hours. No intercostal aneurysms were seen. The patient was given customary amounts of penicillin preoperatively, and penicillin and streptomycin postoperatively. He required only occasional doses of codeine and morphine for chest pain. He did well for the first three days.

On the fourth postoperative day the patient began to complain of diffuse epigastric pain, which was never well localized. Temperature ranged up to 101° F. On the sixth day, penicillin and streptomycin were discontinued and Terramycin was started. On the eighth day, the abdomen became distended. Hyperactive bowel sounds could be heard throughout this period upon auscultation. X-ray films of the chest and abdomen showed no significant

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change. After the sixth day, the leukocyte count remained at 25,000 cells with 85 per cent neutrophils. There was no eosinophilia. Blood cultures were repeatedly negative. The blood pressure had stabilized at about 150/100, and the femoral pulses were strong. A tentative diagnosis of acute appendicitis was entertained, but laparotomy was postponed because there were no localizing signs.

On July 11, 1953, the fifteenth postoperative day, the patient suddenly became pale, dyspneic, and restless. The blood pressure fell and he lost consciousness. He was given plasma, Levophed Bitartrate and oxygen, and an emergency laparotomy was immediately performed. Massive hemoperitoneum was found, with arterial blood entering through the foramen of Winslow. It was thought, in the absence of other evident source, that aortic dissection and rupture had occurred, and the chest was reopened. The aortic anastomosis was found intact. With extension of the abdominal incision, the source of the bleeding was found to be an aneurysmal dilatation of the splenic artery which had ruptured. This was excised with removal of the spleen. On first exploration of the abdomen, it had been noted that approximately three-fourths of the small intestine was discolored and nonviable. Multiple nodular thrombosed vessels were seen in the mesentery. Approximately 300 cm. of involved intestine was now resected with end-to-end anastomosis between jejunum and ileum. The remaining bowel had a normal color although a few nodular lesions remained in the mesentery.

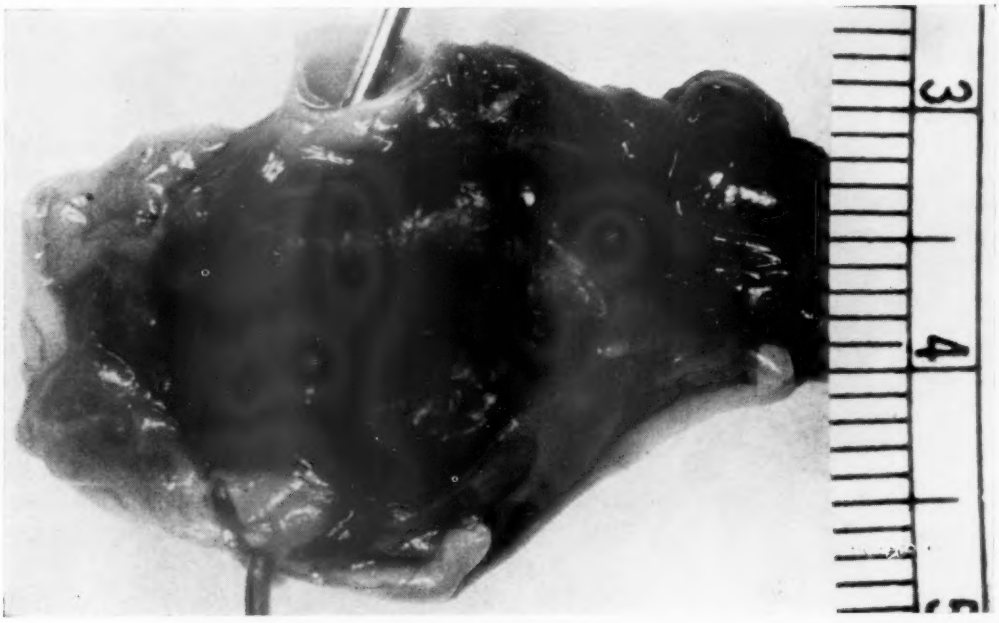

Fig. 1.—Photograph of a typical aneurysm of an intercostal artery found at autopsy in Case 1. This was located lateral to the angle of the rib. Probe is in the lumen.

Postoperatively, blood pressure again became measurable, and the patient was continued on penicillin and streptomycin with aureomycin added. Heparin was also given, but discontinued after six days because of bloody diarrhea and a fall of hemoglobin levels. Adrenocorticotrophic hormone was also begun on July 16, but on July 18 all medications were discontinued. On July 19, 1953, there was extreme respiratory distress and cyanosis which did not respond to treatment, and the patient expired with signs of pulmonary edema twenty-three days after the coarctation repair and eight days after the emergency laparotomy.

Autopsy Findings.—At autopsy there was atelectasis of the left lung and severe edema of the right, with moderate bilateral hemothorax. The heart

weighed 300 grams and the aortic valve was bicuspid. The aortic anastomosis was smoothly healed with no thrombosis or narrowing. The right subclavian artery was enlarged. The intercostal arteries originating below the level of the anastomosis contained thrombosed aneurysms up to 2 cm. in size (Fig. 1). These were lateral to the angle of the rib, in contrast to those occasionally found close to the aorta in long-standing cases of coaretation, and they had an inflammatory appearance. The remaining mesentery contained nodules up to 1 cm. in size which appeared to be thrombosed vessels and small aneurysms. The intestinal anastomosis was healing and the remaining small intestine, measuring 70 cm. in length, had a normal appearance. The liver contained a yellow softened area in the left lobe, beneath which a prominent dilated vessel was occluded by adherent clot. The remainder of the gross findings were not significant.

CASE 2.—The second case is that of a 46-year-old white male who had been hospitalized in state institutions continuously since childhood because of mental retardation. In February, 1953, resection of a segment of the right upper lobe of the lung had been carried out under Pentothal Sodium anesthesia for tuberculosis at Anoka State Hospital, Anoka, Minn. The patient was noted to have blood pressure in the arms ranging up to 200/100, with absent femoral pulses. There was enlargement of the left ventricle with an apical systolic murmur.

The conclusion was reached that he was suffering from a coaretation of the aorta, and on May 6, 1953, the coaretation was resected and an end-to-end anastomosis was made under Pentothal Sodium anesthesia. Penicillin, streptomycin, and isoniazid, which he had been receiving previously, were continued postoperatively. The immediate postoperative period was uneventful.

On the eighth postoperative day the patient developed a low fever and vomited once. By the tenth day the abdomen was distended, tender, and rigid. Temperature rose to 103° F. Abdominal exploration was carried out, and free pus was found in the peritoneal cavity. Gangrenous patches were present along the ileum and distal part of the jejunum. Thirty-eight centimeters of the small intestine was resected. The vessels of the mesentery were not remarkable grossly.

Postoperatively the patient has done well except for some diarrhea, and at the time of writing he is in good physical condition, although still hospitalized at Anoka State Hospital.

MICROSCOPIC DESCRIPTION

Sections of the operative specimen of Case 1 showed the muscular arteries of the mesentery to contain areas of acute necrosis and inflammation. The muscular media showed fraying and disintegration of fibers with infiltration by leukocytes and mononuclear inflammatory cells. The internal elastic lamina frequently was preserved until the media was nearly destroyed. Collections of fibrinoid material were seen in the intima and media. The intima was frequently covered by fresh or partially organized thrombus. Polymorphonuclear leukocytes were found infiltrating the degenerating media and in the adventitia. There was some proliferation of adventitial fibrous tissue, and this contained large numbers of lymphocytes, plasma cells, and macrophages. Eosinophils were infrequently seen. In the dilated segments, the arterial wall seemed to be composed only of adventitial fibrous tissue. The

necrosis of the arterial wall was often found to involve only a portion of the circumference, leaving the remaining wall intact. The smallest lesions appeared to involve the inner part of the media only (Fig. 2).

Fig. 2.

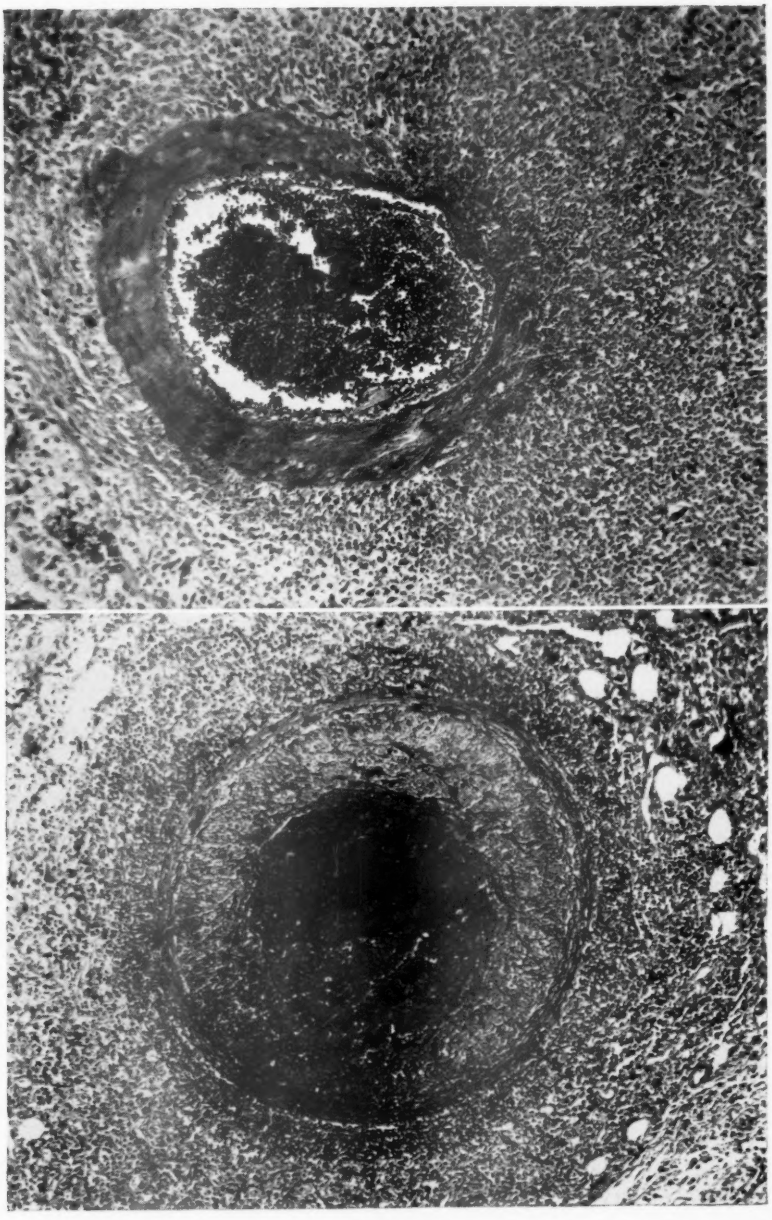

Fig. 3.

Fig. 2.—Photomicrograph of a small mesenteric artery in the surgical specimen of Case 1. Note the patch of necrosis involving only a portion of the arterial wall. (Hematoxylin and eosin stain. ×85.)

Fig. 3.—Photomicrograph of a small mesenteric artery from the surgical specimen of Case 2. The exudate and necrosis involve the entire circumference, and the lumen contains thrombotic material. (Hematoxylin and eosin stain. ×85.)

In the small arteries within the bowel wall, the changes were less intense, with less necrosis and only small collections of chronic inflammatory cells in the adventitia. Sections of the splenic artery in the region of the ruptured aneurysm showed necrosis of the wall similar to that seen in the muscular arteries of the mesentery.

Sections of the autopsy material in this case showed similar necrosis and inflammation in the mesenteric, hepatic, and intercostal arteries. Other arteries were not affected, and no arteries originating above the coarctation were involved. Sections of the liver showed a progressive infarction. There was central lobular ischemic necrosis throughout the liver, with some bile duct proliferation. Sections of the lung showed inflammatory exudate and edema fluid in the alveoli. Sections of the kidney and other organs showed nothing of note.

Examination of the histologic sections of the surgical specimen of Case 2 showed similar changes in the mesenteric arteries. Necrosis of the media with infiltration of the wall by inflammatory cells was found. The involvement of only portions of the arterial walls was again seen. The entire inflammatory process appeared to be less extensive than in the first case. Relatively more polymorphonuclear leukocytes appeared in the adventitial exudate, with few eosinophils. Small amounts of fibrinoid material were found in the necrotic vessels. Aneurysmal dilatation of the arteries was not marked, and there appeared to be less fibrous proliferation (Fig. 3).

The histologic differences seen in the two cases might represent a difference in time interval from the coarctation repair to the resection of the bowel. This interval was fifteen days in the first case and ten days in the second. No evidence of pre-existing arterial disease was found in either case.

COMMENT

It has been noted that the lesions of periarteritis nodosa seldom affect vessels having a low intravascular pressure, such as the pulmonary arteries,[4] and the veins generally. Persons having elevated pressure in the pulmonary arteries may develop lesions,[5] and they may be produced in arterialized veins.[6] Many of the experimental means used to produce lesions resembling those of human periarteritis nodosa in animals involve the marked elevation of the blood pressure.[2, 4, 7] Vessels protected from increased intravascular pressure do not develop necrotic changes.[7]

There is much evidence that lesions of necrotizing arteritis may be produced by methods involving hypersensitivity.[8] Zeek, Smith, and Weeter attempted to distinguish the lesions of hypersensitivity from those of periarteritis nodosa proper, which they stated is primarily related to intravascular pressure and strain.[4] Hopps and Wissler[9] have suggested that arterial spasm in hypersensitivity reactions may cause localization of the lesions at similar points of strain.

Although both of the patients reported here received antibiotics and other medications, there was no overt evidence of hypersensitivity at any time in

the clinical course. The onset of the abdominal pain in the immediate postoperative period in both cases seemed to relate the changes directly to events of the surgery. The most striking event affecting the vascular system was the sudden introduction of pulsatile elevated systemic blood pressure to that portion of the circulation which had never previously been subjected to that stress. It seemed significant that the vascular lesions in the autopsy case, and, as far as can be determined, in the second case also, were limited to those vessels which originated distal to the coarctation repair.

The lesions of periarteritis nodosa in a normal circulatory system may also be limited to vessels of certain organs or regions.[10] The distribution of the lesions in the present cases corresponded to those classified by Zeek[4] as periarteritis nodosa rather than to those considered to be hypersensitivity angiitis. The histologic appearance and distribution of the lesions was not that of the arterial and arteriolar necrosis seen typically in malignant hypertension.

Sealy[11] has mentioned a case clinically similar to those reported here. At autopsy, lesions of a "peculiar thrombosing vascular disease confined to the body area below the coarctation" were found.

The sudden increase in intravascular strain appears to have been the precipitating influence in producing the arteritis in these cases. It is evident that the changes in arterial blood pressure after coarctation repair must be accompanied by an abnormal response of the patient to produce a necrotizing arteritis. Hypersensitivity may be one factor in this altered response.

SUMMARY

Two cases of necrotizing panarteritis indistinguishable from periarteritis nodosa occurring immediately after surgical repair of coarctation of the aorta are presented. The lesions produced infarction of the intestine in both cases, with death of one patient. The lesions at autopsy were limited to the arteries supplied by the aorta below the coarctation. The possible significance of the sudden increase in blood pressure in precipitating the lesions of periarteritis nodosa is discussed.

We wish to acknowledge the assistance of Dr. F. John Lewis of the Department of Surgery, and of Dr. E. G. Evans of the Department of Pathology, who performed the autopsy reported.

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VENOUS THROMBOSIS OF THE UPPER EXTREMITY COMPLICATED BY PULMONARY EMBOLUS

Case Report

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VENOUS thrombosis of the upper extremity is a relatively uncommon condition, constituting but a small percentage of all cases of venous thrombosis. Barker and his associates,[1] in a review of 1,260 cases of thrombosis, found that in only 1.7 per cent of the patients did thrombosis originate in the upper extremity. DeCamp and his associates,[3] in reporting a group of ninety cases of "spontaneous thrombophlebitis," cited only one case in which the upper extremity was involved. Because of the unusual onset of symptoms and because of the many problems involved in management, the following case of thrombosis of the left axillary vein complicated by pulmonary embolism is reported in detail.

CASE REPORT

A 35-year-old Negro man (Reg. No. 410-11) was admitted to the 320th General Hospital, A.P.O. 180, on Jan. 11, 1953, with the chief complaint of pain and swelling of the left arm. Four days prior to admission, while on board ship en route to the European theater, the patient noted the onset of tingling pain in the left arm and fingers. A history of trauma could not be elicited. At this time there was no restriction of arm motion. The day before admission, the left arm and hand suddenly became swollen and painful, and he could not flex the fingers or elbow. He had no chills or fever; there was no cough, sputum, or chest pain. The patient's past history and system review did not contribute significantly to the present illness.

Physical examination showed a muscular Negro man in moderate discomfort, with an oral temperature of 98.6° F.; the pulse rate was 80 per minute; respirations, 20 per minute; and the blood pressure, 120 mm. systolic and 80 mm. diastolic. Positive findings were limited to the neck and left upper extremity. The left side of the neck was edematous, and both the supra- and infraclavicular fossae were markedly swollen, but without palpable masses or tenderness. There was massive swelling of the left upper extremity from shoulder girdle to finger tips, with numerous dilated veins over the upper arm and shoulder girdle; the cephalic vein was distended. There was no localized tenderness, nor was there any axillary adenopathy. The left radial pulse was strong and equal to that on the right. The fingers were warm. All arm and hand movements were painful because of swelling. The physical examination was otherwise normal.

Laboratory studies on admission showed the hemoglobin to be 13.2 Gm.; the red blood cell count was 3,950,000; the white blood cell count was 7,100, with neutrophils 66 per cent, lymphocytes 26 per cent, monocytes 2 per cent, and eosinophils 6 per cent. Urinalysis was negative. Cardiolipin was negative. A roentgenogram of the chest taken on admission was within normal limits.

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On admission it was felt that the patient had a thrombosis of the left axillary vein, and the early treatment consisted of elevation and warm packs. During the first two and one-half days of hospitalization, the patient was fairly comfortable and afebrile; the swelling of the hand and forearm decreased remarkably, but the enlargement of the upper arm persisted.

At 10 P.M. on the third hospital day, the patient suddenly complained of severe, unremitting pain, pleuritic in nature, in the left lower chest and the left upper quadrant of the abdomen, associated with the onset of a nonproductive cough. There was no sputum or hemoptysis. He became extremely dyspneic and apprehensive, and thought that he was dying. Temperature was 99° F. orally; the pulse rate, 88 per minute; and the blood pressure, 180 mm. systolic and 70 mm. diastolic. He appeared to be in severe discomfort. The chest showed splinting of the left side with shallow respirations. There were persistent râles in the left lower chest laterally. There was no friction rub. The white blood cell count was 9,400, with 66 per cent neutrophils. Ten hours after the onset of pain, a roentgenogram of the chest was normal. The clinical impression was that of pulmonary embolus of the lower lobe of the left lung.

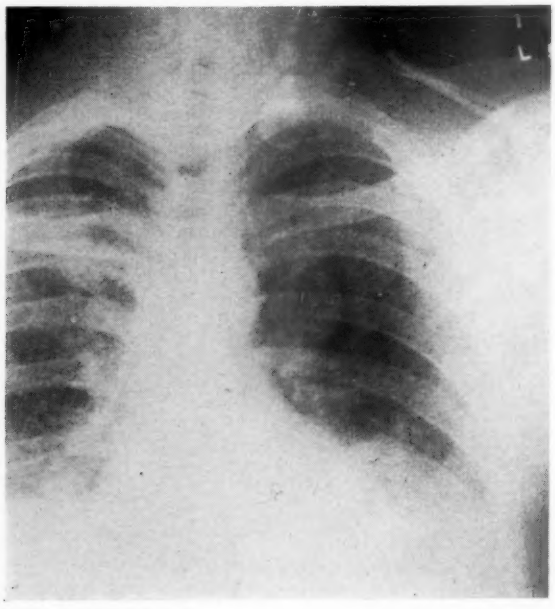

Fig. 1.—Roentgenogram revealing a definite infiltration in the lower lobe of the left lung.

At this time, ten hours after the onset of pain, anticoagulation therapy was begun, using both heparin and Dicumarol. The heparin was discontinued after seventy-two hours, when an effective Dicumarol level was attained. During the succeeding three days, the patient remained acutely ill, with severe chest and abdominal pain, dyspnea, and tachycardia. He was placed in an oxygen tent, and notable relief was obtained.

Sixty hours after the onset of pain, bronchial breathing was noted in the left axillary line anteriorly. At this time, a roentgenographic examination revealed a definite infiltration in the lower lobe of the left lung (Fig. 1). Temperature rose to 101° F. orally, and then returned to normal within seven days.

Eight days after admission (six days after embolization) the administration of oxygen was discontinued. Steady improvement followed, although the patient occasionally complained of pain in the lower left side of the chest, without objective evidence of further embolic phenomena. The swelling of the arm gradually decreased; however, the shoulder

girdle and the clavicular fossae remained edematous, and evidence of collateral circulation was manifest throughout the hospital stay. Dicumarol therapy was continued for five and one-half weeks; a prothrombin level of from 11 to 35 per cent of normal was maintained.

On the twenty-fourth hospital day, infrared photographs were obtained which demonstrated persistence of the collateral circulation over the shoulder girdle (Fig. 2). The thickened axillary vein could be readily palpated at this time.

Fig. 2.—Photograph demonstrating persistence of the collateral circulation over the shoulder girdle.

Fig. 3.—Phlebogram taken with the arm in the horizontal position showing no dye passing through the axillary vein proximal to the axilla.

On the forty-sixth hospital day, phlebograms were taken with the arm at the side. Injection of 20 c.c. of Neo-Iopax demonstrated a block of the axillary vein deep in the axilla, with collateral circulation passing over the shoulder girdle and down the long

thoracic vein, entering the intercostal vessels. Phlebograms were repeated eight days later with the arm in the horizontal position, and again showed no dye passing through the axillary vein proximal to the axilla (Fig. 3). Following phlebography, the patient experienced pain and discomfort in the arm, associated with thrombosis of the antecubital veins at the site where the dye was injected. This responded rapidly to warm applications.

Eight weeks after admission, the patient was asymptomatic, and the only abnormal physical findings were the palpable axillary vein, the dilated collateral vessels, and the thrombosed antecubital veins which had been noted previously. It was felt that this man was not qualified for duty in an overseas theater, and he was therefore returned to the United States for further treatment and disposition.

DISCUSSION

Most cases of upper extremity thrombosis generally follow either mild repetitive trauma, congestive heart failure, or obstructing neoplasm.[8, 9] In the case presented here, we were unable to establish an etiology; there was no history of trauma, and there was no evidence of either cardiac failure or neoplasm. It may well be that this represents a case of so-called "spontaneous thrombophlebitis."

The diagnosis of axillary vein thrombosis was made on admission. Anticoagulants were not administered immediately because it was felt that pulmonary embolus rarely, if ever, occurs in this type of thrombosis. In 1949, Hughes[4] reviewed 320 reported cases of upper extremity thrombosis and concluded that no case of this syndrome had ever ended fatally. He stated that "it is doubtful whether nonfatal pulmonary embolism has ever occurred." Kleinsasser[5] likewise was of the opinion that there was practically no tendency to embolization in effort thrombosis.

In a study of fifty-eight cases of axillary and subclavian vein thrombosis reported by Veal and Cotsonas,[8] there were seventeen cases of pulmonary embolism. All emboli occurred, however, in stasis thrombosis secondary to congestive heart failure. Barnett and Levitt,[2] in 1951, described what they believed to be the first reported case of pulmonary embolism following effort thrombosis of the axillary vein. Tomlin[7] later re-emphasized the serious nature of thrombophlebitis of the upper extremity by presenting in detail five cases complicated by pulmonary embolism. Wright[9] has stated that the incidence of pulmonary embolus is lower in upper extremity thrombosis than in other types of thrombosis, but that it does occasionally occur. It would seem, therefore, that anticoagulants should be included in the initial therapy of upper extremity thrombosis in an attempt to prevent embolization.

The prognosis following upper extremity thrombosis is relatively poor. Residual fatigue is common, even after the subsidence of edema[6]; and repeated attacks of thrombosis are prone to occur.[9]

SUMMARY AND CONCLUSIONS

1. A case of venous thrombosis of the left upper extremity complicated by pulmonary embolus is presented in detail.

2. Pulmonary embolus complicating upper extremity thrombosis occurs more frequently than is supposed, and this fact must be borne in mind.

3. Anticoagulant therapy is recommended in the early treatment of all cases of upper extremity thrombosis.

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Editorial

Biologic Behavior of Carcinoma of the Lung

WHATEVER the predisposing and inciting causes may be, carcinoma of the lung is on the increase. It is more common in the male than in the female and occurs more often in the white person than in the Negro. The order of frequency is white male, Negro male, white female, and Negro female. Irrespective of sex or race, the age incidence appears to be the same, most commonly the sixth decade of life.

All carcinomas of the lung originate in the mucosa of the bronchial tree near a bifurcation. The cells lining the alveoli are not likely to produce a carcinoma of the lung in man. About one-half of the carcinomas are located in a main bronchus, often close to the bifurcation of the trachea, or near the opening of a bronchial branch that leads to a lobe of the lung. The remainder originate more peripherally in a bronchial branch to a lobe or in a bronchial twig to a segment of a lobe. From the bronchial mucosa where it originates, the carcinoma grows outward away from the bronchial wall. Usually, at first it does not ulcerate or protrude into the lumen nor does it encircle the wall of the bronchus. The growth expands away from the bronchial wall and infiltrates the near-by tissue, lymph, and blood spaces. Before the carcinoma protrudes into the lumen and obstructs it, a considerable portion of the lung tissue adjacent to the bronchus is already involved.

Like other carcinomas, those of the lung infiltrate locally and metastasize to regional lymph nodes and to other organs. The over-all cellular pattern of each carcinoma of the lung as it exists throughout the growth is usually reproduced in all the distant sites to which the cells are transported and where they become implanted. A small primary growth may produce a distant metastasis many times its own size. Distant metastases from carcinoma of the lung have a predilection for the liver, the suprarenal glands, and the brain, in that order. Metastasis to the brain occurs in one of three patients with carcinoma of the lung. The cell type bears no relation to the frequency of intracranial metastasis.

The biologic features just enumerated appear to be common to all carcinomas of the lung. This might be expected, since all carcinomas of the lung, whatever their cellular structure, originate from the same ancestor cell, the reserve cell. The reserve cells, that appear to be the parent cells of all carcinomas of the lung, are at the bottom of the epithelial cell layer lining the bronchial tree. They are the only epithelial cells here still capable of multiplication. The reserve cells replenish the columnar and goblet cells when they

are spent or injured. In the process of cell division, and presumably under the influence of some carcinogenic agent, one or more reserve cells acquire malignant properties and become cancer cells. Under such circumstances the reserve cell may differentiate into cancerous columnar cells or cancerous squamous cells, or it may multiply into undifferentiated cancer cells. Accordingly, carcinomas of the lung may be grouped into columnar cell carcinomas, squamous cell carcinomas, and undifferentiated, that is, reserve cell carcinomas. About 50 per cent of the carcinomas of the lung are squamous cell carcinomas, less than 30 per cent are reserve cell carcinomas, and less than 20 per cent are columnar cell carcinomas. The rest are mixed columnar cell and squamous cell carcinomas, with occasionally all three cell types represented in the same growth. While multicentric origin of carcinoma of the lung has been observed, two carcinomas of the lung, each having a different microscopic structure, rarely occur in the same individual.

Within each histologic group, there is a range of variations in cellular pattern, degree of differentiation, rapidity of growth, and the amount and kind of stroma induced. Regardless of the cell type, carcinoma of the lung may grow slowly or rapidly. The speed of growth is apparently governed by factors other than the cellular pattern.

It is as yet undetermined under what influence or influences the dividing reserve cell gives rise to cancer cells. If the reserve cell that divides into cancer cells has progressed toward differentiation into columnar cells, the carcinoma is of the columnar cell type. If the reserve cell divides into cancer cells when its potential to become a squamous cell is dominant, the carcinoma is of the squamous cell type. If the reserve cell divides into cancer cells while it is uncommitted, it reproduces itself, and the resulting carcinoma is of the reserve cell type (that is, undifferentiated). Thus, the cellular pattern appears to depend on the direction and the degree of differentiation of the reserve cell at the time it gives rise to cancer cells.

Under physiologic conditions, the reserve cell multiplies for the purpose of replacing the top cells lining the bronchial mucosa. The same factors or agents that accelerate or interfere with the normal multiplication of reserve cells may be responsible for the carcinogenesis. The behavior of the carcinoma thus induced seems to depend not on the particular pattern formed by the cells but on inherent characteristics of the cancer cell. This concept of the cellular origin of carcinoma of the lung may aid in comprehending its biologic behavior.

—Béla Halpert

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—Béla Halpert

Recent Advances in Surgery

Conducted by Alfred Blalock, M.D.

A STUDY OF PATIENTS FOLLOWING TOTAL AND NEAR-TOTAL GASTRECTOMY

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UNTIL recent years, total gastrectomy has been considered an operation justifiable only in cases in which a lesser procedure is not possible. With the great improvements which have attended advances in pre- and postoperative care and in anesthesia, a number of reports[1-3] show that in skilled hands total gastrectomy may be accomplished with no greater hazard than for partial gastrectomy. At best, partial gastrectomy can be considered only a compromise between the principles of cancer surgery and the consequences of total extirpation of the stomach. In view of the more favorable mortality attending total gastrectomy now, several surgeons[1-3] feel that total gastrectomy should be considered in the routine treatment of gastric cancer of all types. Although the survival rates have not been encouraging following total gastrectomy for cancer, much of this is undoubtedly due to the type of case for which this procedure his been reserved.

Another deterrent to the use of total gastrectomy has been the impression that these patients are more likely to have nutritional difficulties of a severe degree than do patients who undergo lesser resections of the stomach.

This study was undertaken to give further information regarding the condition of human beings following total or near-total removal of the stomach.

Material and Methods.—Twenty-two individuals were studied following total gastrectomy which was performed from two months to eight years earlier. Four additional patients were studied who had undergone near-total gastrectomy (more than 90 per cent of the stomach removed) from two to six years earlier. Four patients were women, twenty-two men. The age ranged from 28 to 83 years. Seventeen of the operations were for carcinoma, four for gastric ulcers which proved to be benign, four for gastric polyps, and one for esophageal varices. Three of the patients operated on for cancer were found to have evidence of recurrence at the time of the study. The remainder showed no evidence of recurrent cancer. Following total gastrectomy, esophagoduodenostomy was performed in three instances, esophagojejunostomy plus entero-

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enterostomy in eleven instances, and a Roux Y reconstruction in seven instances. The Hofmeister modification of the Billroth II operation was used in all four cases following near-total gastrectomy. All patients were admitted to the hospital for a period of eight to ten days, during which time the following studies were performed:

1. Routine history and physical examination with special inquiry into the patients' eating habits and gastrointestinal symptoms.

2. Determination of hemoglobin, erythrocytes, leukocytes, and differential, mean cell diameter, reticulocytes, blood urea nitrogen, and total serum protein. In some cases, blood CO_2 and chloride were determined.

3. Roentgenologic examination with barium of the esophagus and upper small bowel, as well as estimation of the transit time to the cecum in some cases.

4. Measurement of fecal fat and nitrogen excretion. Three- to four-day collection periods were used with carmine dye orally to mark the interval. A weighed diet containing 145 Gm. of carbohydrate, 80 Gm. of fat, 80 Gm. of protein with a total of 1,620 calories was offered, and the amount eaten calculated. Fecal fat was determined by the method of Fowweather[4] and fecal nitrogen by the method of Kjeldahl.[5] Fat excretion in the stool was usually reported in percentage of the ingested fat. Fecal nitrogen was reported in grams per day excreted.

5. Pancreatic function was studied by aspirating the intestinal content a short distance below the anastomosis of the esophagus to the intestine. In some instances two intestinal tubes were inserted under fluoroscopic control. One of these tubes had a balloon near the tip just proximal to the opening in the tube. This tube was positioned fluoroscopically so that it lay in the efferent jejunal loop a short distance below the enteroanastomosis. This was then inflated with about 30 to 40 c.c. of air so that the duodenal content would be dammed back and could be aspirated by the second tube placed just proximal to it. In addition, various stimuli could be injected through the distal tube into the efferent loop of jejunum, simulating the conditions present on eating but preventing contamination of the duodenal content. The volume, pH, amylase, lipase, and trypsin, and in some instances bicarbonate, were measured. The pH was determined with a glass electrode. Amylase was determined by the method of Somogyi,[6] trypsin by the method of Dorothy Anderson,[7] and lipase by a modification of the method of Cherry and Crandall.[8] Specimens were collected on ice and frozen until the determination was done.

6. Iron absorption: Serum irons were determined by the method of Barkan and Walker.[9] Iron absorption was studied by first drawing a fasting blood level, then administering 1.0 Gm. of ferrous gluconate orally, and drawing blood specimens at one-, three-, and five-hour intervals.

7. Esophagoscopy: Eleven individuals were examined by esophagoscopy at different times following total gastrectomy by an experienced esophagoscopist. The procedure was performed under general anesthesia.

Results.—The results are tabulated in detail in Tables I through VIII. The essential points may be summarized as follows:

TABLE I. WEIGHTS AFTER TOTAL AND NEAR-TOTAL GASTRECTOMY

PATIENT	AGE (YR.)	SEX	DIAGNOSIS	TYPE OF ANASTOMOSIS	NORMAL PREOPERATIVE WEIGHT	WEIGHT IMMEDIATELY POSTOPERATIVELY	PRESENT WEIGHT	INTERVAL FROM SURGERY	% OF WEIGHT LOSS FROM NORMAL
				Total Gastrectomy					
1. A*	69	M	Ca	E-D†	170	150	108	4 mo.	37
2. B	67	M	Ca	Roux Y	160	132	130	4 mo.	19
3. C	70	F	Ca	E-J‡	155	143	123	4 mo.	21
4. D	35	M	Ca	Roux Y	195	176	140	4½ mo.	28
5. E	28	F	Esophageal varices	E-J	127	118	90	5 mo.	24
6. F	75	M	Ca	E-J	200	190	152	5 mo.	24
7. G	64	M	Ca	Roux Y	140	118	103	7 mo.	27
8. H	46	F	Ca	Roux Y	120	115	93	8 mo.	22.5
9. I	60	M	Gastric polyps	E-D	215	200	140	8 mo.	35
10. J	71	M	Ca	Roux Y	165		115	9 mo.	36
11. K	67	M	Ca	E-D	200	158	154	10 mo.	23
12. L	63	M	Ca	E-J	165	140	110	14 mo.	33
13. M	74	M	Gastric ulcer	Roux Y	145	119	112	17 mo.	22.7
14. N	79	M	Gastric polyps	E-J	160	149	125	22 mo.	22
15. O	83	M	Ca	Roux Y	138	84	114	24 mo.	17.3
16. P	66	M	Ca	1st operation subtotal, Billroth II	130	116	115	26 mo.	11.5
				2nd operation total, E-J	115	104	88	14 mo.	20.8

17. Q	69	M	Gastric ulcer	E-J	160	137	133	31	mo.	16.9
18. R	68	M	Ca	E-J	170	158	138	36	mo.	25
19. S	68	M	Ca	E-J	177	115	120	41	mo.	32
20. T	28	F	Gastric polyps	First operation subtotal, Billroth II	155		145	5	yr.	6.4
				Second operation total, E-J	145		135	2	mo.	12.8
21. U	70	M	Gastric ulcer	E-J	170	149	154	6	yr., 4 mo.	15.3
22. V	68	M	Ca	E-J	145	130	116	8	yr., 9 mo.	20
					Near-total Gastrectomy					
1. W	72	M	Gastric polyps	Billroth II	152	132	118	2	yr.	22
2. X	46	M	Gastric ulcer	Billroth II	158		138	4½	yr.	13
3. Y	66	M	Ca	Billroth II	130	115	110	6	yr.	15
4. Z	65	M	Ca	Billroth II	130	128	132	6	yr.	0

Total gastrectomy: All cases average weight loss = 24 per cent; median weight loss = 23 per cent. Less than one year postoperative average weight loss = 27 per cent; median weight loss = 24 per cent. More than one year postoperative average weight loss = 22 per cent; median weight loss = 22 per cent.

Near-total gastrectomy: Average weight loss = 12.5 per cent; median weight loss = 14 per cent.

*One-half of pancreas excised.

†E-D = Esophagoduodenostomy.

‡E-J = Esophagojejunostomy with enteroanastomosis.

1. *Weight change:* The figures for twenty-two patients after total gastrectomy show an average weight loss of 24 per cent (median value = 23 per cent loss) compared to normal preoperative weight. The range was from 12.8 per cent to 37 per cent loss. One-half of this group had survived operation by less than one year at the time of observation and the remainder had survived for one year or more. The average weight loss in the former group was 27 per cent with a median value of 22 per cent. Although the number of patients is small, there is no particular difference in weight loss following total gastrectomy when the patients are compared on the basis of the type of anastomosis performed.

The four cases studied after near-total gastrectomy showed an average weight loss of 12.5 per cent with a median value of 14 per cent.

2. *Eating habits and gastrointestinal symptoms:* Fifteen of eighteen patients eat more frequent meals than normal. None of the patients felt they were able to eat as much at one time as they normally had prior to the onset of illness. The average intake at one meal was about two-thirds of the preoperative normal quantity. There was considerable variation in this regard, the quantity intake ranging from 20 to 25 per cent up to 90 to 95 per cent of the normal quantity.

TABLE II. GASTROINTESTINAL SYMPTOMATOLOGY

PATIENT	POSTOPERATIVE INTERVAL	NUMBER OF MEALS* PER DAY	QUANTITY† OF INTAKE (%)	DYSPHAGIA‡	NAUSEA OR VOMITING‡	DISTRESS OR PAIN‡	WEAKNESS‡
1. A	4 mo.	3 M, 3 S	50	+	0	0	1+
2. B	4 mo.	3 M, 2 S	50	0	0	1+	2+
3. C	4 mo.	6	33	0	0	2+	0
4. D	4½ mo.	5	50	0	0	+	0
5. E	5 mo.			0	2+	1+	2+
6. F	5 mo.	4-5	50	+	0	0	1+
7. G	7 mo.			0	1+	1+	1+
8. H	8 mo.	6	50	+	1+	0	2+
9. I	8 mo.			0	1+	1+	2+
10. J	9 mo.			0	1+	2+	3+
11. K	10 mo.	4	85	0	0	0	0
12. L	14 mo.		20-30	+	0	1+	2+
13. M	17 mo.			0	0	1+	1+
14. N	22 mo.	3-4	90-95	0	0	0	0
15. O	24 mo.	3 M, 2 S	85	0	0	0	1+
16. P	26 mo.	Numerous	25	+	1+	1+	3+
17. Q	31 mo.	3	66-75	Occasional	0	0	0
18. R	36 mo.	Very frequent	Small amounts	+	1+	1+	3+
19. S	41 mo.	8	66	Slight	0	0	1+
20. U	6 yr., 4 mo.	3 M, 3 S	90	0	0	0	1+
21. V	8 yr., 9 mo.	3	90-95	0	0	0	0
1. W	2 yr.	6	50	0	0	1+	1+
2. X	4½ yr.	3 M, 2 S	75	0	0	0	0
3. Y	6 yr.	3 M, 1 S	75	0	0	0	0
4. Z	6 yr.		80	0	0	0	0

*M = meal, S = snack.
†Normal meal when well = 100 per cent.
‡0-4 + scale.

Dysphagia was a complaint in eight patients. In one patient it was present only occasionally, and in a second patient the difficulty was very slight.

Three patients complained of mild difficulty in swallowing and three experienced moderate to severe difficulty. All of the patients except one who complained of dysphagia had roentgenologic evidence of narrowing at the anastomosis roughly proportional to the severity of the complaint. The one patient who was negative radiographically had very mild dysphagia of an intermittent type.

Of the three cases of moderate to severe dysphagia, two were apparently due to benign stricture and one to recurrent carcinoma.

Mild distress in the epigastrium and nausea and/or vomiting were present in about one-third of the entire group after meals. Weakness was present in nearly two-thirds of the group, being severe in three of twenty-two patients.

3. *Fecal fat and nitrogen excretion:* Fourteen patients with a total gastrectomy were studied and the value reported in terms of percentage of the ingested fat which was excreted in the stool. The figures range from 5.8 to 20.5 per cent with a mean value of 11.5 per cent on a diet containing 85 Gm. of fat per day. Normal values for fat excretion in the feces are given by various authors[10-12] as from 5 to 10 per cent of the dietary intake. Taking 10 per cent as the upper limit of normal, only eight of the fourteen patients showed an increased excretion of fat. Using 5 per cent as the upper limit of normal, all fourteen showed an increased excretion of fat. Two additional cases showed values of 4.9 Gm. and 2.7 Gm. of fecal fat per day. Both of these may be considered normal.

TABLE III. FAT AND NITROGEN EXCRETION AFTER TOTAL AND NEAR-TOTAL GASTRECTOMY

PATIENT	AGE (YR.)	SEX	POSTOPERATIVE INTERVAL	TYPE OF ANASTOMOSIS	FAT*	NITROGEN (GM. PER DAY)
			Total			
1. A	69	M	4 mo.	E-D	11.6%	0.7
2. B	67	M	4 mo.	Roux Y	7.0%	1.2
3. C	70	F	4 mo.	E-J	5.8%	0.7
4. D	35	M	4½ mo.	Roux Y	15.5%	0.2
5. E	28	F	5 mo.	E-J	4.9 Gm. per day	1.1
6. G	64	M	7 mo.	Roux Y	9.0%	0.5
7. H	46	F	8 mo.	Roux Y	10.8%	0.4
8. I	60	M	8 mo.	E-D	12.0%	1.75
9. J	71	M	9 mo.	Roux Y	8.7%	1.17
10. L	63	M	14 mo.	E-J	2.7 Gm. per day	0.7
11. M	74	M	17 mo.	Roux Y	20.5%	0.3
12. O	83	M	24 mo.	Roux Y	12.2%	0.2
13. Q	69	M	31 mo.	E-J	13.3%	2.1
14. R	68	M	36 mo.	E-J	33% dry wt.	1.2
15. T	28	F	5 yr.	E-J	6.0%	0.5
16. U	70	M	6 yr., 4 mo.	E-J	20.4%	1.0
17. V	68	M	8 yr., 9 mo.	E-J	8.6%	1.6
			Near-total			
1. W	72	M	2 yr.	Billroth II	3.6%	1.49
2. X	46	M	4½ yr.	Billroth II	1.5%	1.34

*Percentage of ingested fat excreted in feces, unless otherwise indicated, with dietary intake of 80 Gm. of fat per day.

Two patients with near-total gastrectomy both show values of less than 5 per cent excretion of dietary fat. Therefore, of sixteen patients with total or near-total gastrectomy, only eight showed mildly increased fecal excretion

TABLE IV. PANCREATIC FUNCTION STUDIES

PATIENT	POSTOPERATIVE INTERVAL	TYPE OF SPECIMEN	VOLUME (C.C.)	pH	MEQ. PER L. OF HCO_3	AMYLASE (SOMOGYI UNITS)	LIPASE (C.C. OF 0.05N NaOH)	TRYPSIN (TUBES LIQUEFIED)
		After Total and Near-total Gastrectomy						
1. B	4 mo.	Fasting	0.2	7.0	0	103,600	0.0	0
		Fasting	4.5	7.45	0	151,500	0.1	5-9
		Secretin	25	7.36	0	75,150	0	6-9
		Secretin	11	8.3	0	46,300	0	6-9
		Secretin	17.0	7.7	0	77,250	0.3	6-9
2. C	4 mo.	Fasting	16	7.5	8.9	39,200	0.4	5-9
		Milk	31	6.9	0	40,500	0.2	5-9
		Eggnog	45	7.0	13.7	13,500	3.5	6-9
		0.1N HCl	105	7.1	8.9	9,900	0.3	6-9
3. G	7 mo.	Fasting	8.0	7.55	0		0.2	6-9
		Fasting	15.0	7.36	0		0.6	5-9
		Secretin	4.0	7.11	0		Insufficient quantity	5-9
		Secretin	8.0	8.05	2.0		0.1	6-9
		Secretin	15.0	7.64	3.0		0.1	6-9
4. I	8 mo.	Fasting	17.5	7.56	14.4	29,800	0.3	4-9
		Fasting	47.0	7.01	12.3	28,000	0.4	5-9
		Secretin	50.0	7.64	38.8	27,000	0.5	5-9
		Secretin	97.0	7.69	29.7	35,500	0.4	6-9
		Secretin	35.0	7.28	21.5	35,800	0.2	4-9
5. J	9 mo.	Fasting	12.0	7.8	3	30,100	0.2	8-9
		Fasting	22.0	7.6	0	45,800	0.7	5-9
		Secretin	2.0	8.0	0	33,200	0	0
		Secretin	27.0	7.7	30	51,800	0.1	8-9
		Secretin	12.5	8.0	22	55,400	0	5-9
6. L*	14 mo.	Fasting				53,600	0	5-9
		Skim milk				Insufficient quantity	0.2	6-9
		0.1N HCl				73,400	0	4-9
		Secretin				38,600	0	4-9
7. N	22 mo.	Fasting	5.0	Insufficient quantity		Insufficient quantity	0.1	7-9
		Skim milk	8.0	7.65		14,050	0.1	5-9
		0.1N HCl	20.0	7.6		15,900	19.8	5-9
8. Q	31 mo.	Fasting	8.0			3,850		0
		Skim milk	17.0	8.2		7,900		5-9
		Eggnog	26.0	8.05		2,400	10.47	8-9
		0.1N HCl	35.0			3,550	22.65	8-9
9. S	41 mo.	Fasting	15	7.6		7,400	0	5-9
		Eggnog	125	7.1		7,000	20.98	4-9
		0.1N HCl	54	7.0		28,100	17.28	5-9
		Secretin	71	7.45		36,900	7.91	5-9
10. T	5 yr., 2 mo.	Fasting	49	7.6		13,700	3.78	3-9
		Skim milk	36	7.05		7,400	0.78	5-9
		Eggnog	52	7.2		8,100	1.75	5-9
		Dilute HCl	88	7.5		7,900	22.0	5-9
		Secretin	47	7.7		9,600	21.6	5-9
11. U	6 yr., 4 mo.	Eggnog	37	6 7	0	11,200	20.98	3-9
		Dilute HCl	71	7.3	0	4,700	13.37	6-9
12. V	8 yr., 9 mo.	Fasting	11	7.5		8,400	0.1	5-9
		Dilute HCl	28	6.35		12,700	12.38	5-9
		Secretin	29	7.2		6,950	12.97	4-9
13. W	2 yr.	Fasting	37	7.75	5.0	48,800	0.44	7-9
		Skim milk	36	7.45	0.3	34,800	0.19	4-9
		Eggnog	68	7.4	0.3	21,800	1.68	2-9
		Dilute HCl	93	7.35	5.0	14,200	0.82	5-9
14. X	4½ yr.	Fasting	20	7.25	0	16,200	0.66	5-9
		Skim milk	16	7.6	5.0	31,600	0	6-9
		Eggnog	35	7.45	5.0	9,600	0.72	6-9
		Dilute HCl	54	8.0	23.0	14,800	0	5-9

TABLE IV—CONT'D

PATIENT	POSTOPERATIVE INTERVAL	TYPE OF SPECIMEN		VOLUME (C.C.)	pH	MEQ. PER L. OF HCO_3	AMYLASE (SOMOGYI UNITS)	LIPASE (C.C. OF 0.05N NaOH)	TRYPSIN (TUBES LIQUEFIED)
15. Y	6 yr.	Fasting		28	7.05		75,800	4.0	4-9
		Eggnog		105	6.98		72,800	19.2	2-9
		Dilute HCl		186	7.55		69,000	0	5-9
		Secretin		105	7.75		52,600	0	5-9
16. Z	6 yr.	Fasting		34	7.2	0	1,800	0	6-9
		Skim milk		27	7.8	4.5	1,800	18.9	4-9
		Eggnog		75	7.2	4.5		19.8	4-9
				Normals					
1. B. M.		Fasting	30′	10	6.8		24,400	0.2	4-9
		After	20′	38	7.1	22	16,200	0.2	4-9
		Secretin	40′	18	7.7	4	11,500	0.2	5-9
			60′	37	7.0	4			6-9
2. F. S.		Fasting	30′	121	2	0	------	---	Negative
		After	20′	44	9	64	20,000	0.2	2-9
		Secretin	40′	117	8	57	12,700	0.05	6-9
			60′	61	7	34	12,900	0.3	6-9
3. M. E.		Fasting	30′	16	6.9	---	11,150	0.2	3-9
		After	20′	20	7.3	45			
		Secretin	40′	7.5	8.0	36	17,450	0.5	3-9
			60′	0.4	8.3	0			
4. K. S.		Fasting	30′	15.5	7.5	0	8,400	---	3-9
		After	20′	13	7.7				
		Secretin	40′	33	7.6	28	15,200	0.38	4-9
			60′	27	8.1				
5. C. L.		Fasting	30′	95	6	10	0	---	0
		After	20′	20	8	12.5			
		Secretin	40′	50	8	21	16,550	---	6-9
			60′	40	8	30			

*One-half pancreas excised.

of fat if 10 per cent is used as the upper limit of normal. Again, there is no significant difference in fat excretion in comparing the three types of anastomosis used.

Protein excretion as measured by the nitrogen content of the feces was not significantly elevated in any of the nineteen patients. Two grams of nitrogen in the feces per day was used as the upper limit of normal.

4. *Pancreatic function* (Table IV): Pancreatic function was studied in sixteen patients. Twelve patients were studied in regard to their response to various stimuli such as skim milk, eggnog, and dilute HCl, as well as secretin in some instances. Four patients had secretin tests only. Five normal individuals were also studied using the secretin test. None of the patients show changes which could indicate a change in pancreatic function.

5. *Hematology* (Table V): Values are given for twenty-two patients after total gastrectomy and for four patients after near-total gastrectomy. Six of the twenty-two (Cases 6, 17, 20, 22, 25, and 26) have previously treated blood abnormalities. One of these (Case 6) was diagnosed as pernicious anemia shortly before a carcinoma of the stomach was discovered. Three patients (Cases 22, 25, and 26) developed severe anemia eight years, five years, and six years postoperatively, respectively. All three cases proved to be megaloblastic anemia on hematologic study. One patient (Case 20) developed megaloblastic anemia fifteen months after subtotal gastrectomy performed for bleeding gas-

TABLE V. BLOOD PICTURE AFTER TOTAL AND NEAR-TOTAL GASTRECTOMY

PATIENT	AGE (YR.)	SEX	INTERVAL SINCE SURGERY	PRE-OPERATIVE HEMOGLOBIN (GM.)	PRESENT HEMOGLOBIN (GM.)	R.B.C. (MILLIONS)	HCT (%)	RETICULOCYTES (%)	MEAN CELL DIAMETER (μ)	MCV (μ³)	MCH (γγ)	MEAN CORPUSCULAR HEMOGLOBIN CONCENTRATION (%)	BUN (MG. %)	CO_2 (MEQ./L.)	Cl (MEQ./L.)	TSP (GM. %)
1. A	69	M	4 mo.	15.0	14.4	5.22	44	1.0	7.4	84	27.6	32.7				
2. B	67	M	4 mo.	13.6	12.1	3.62	38.5	0.3		106	34	31	11	24	106	5.9
3. C	70	F	4 mo.	10.4	13.5	3.75	43	1.0	8.0	114.7	36	31.4	10	27	113	6.5
4. D	35	M	4½ mo.	11.7	15.0	5.36	47	1.4	7.2				22			6.6
5. E	28	F	5 mo.	9.4	14.0	4.87	44						13	25	107	6.0
6. F	75	M	5 mo.	9.9	13.4	4.18	41	0.6	7.8	99	30.6	33.3	24			7.0
7. G	64	M	7 mo.	9.8	11.8	4.26	39	1.0	7.3	91.5	27.7	30.3	32	28	105	6.6
8. H	46	F	8 mo.	14.8	13.2	4.36										
9. I	60	M	8 mo.	15.7	14.5	4.33				102	34	33	12	30	104	6.2
10. J	71	M	9 mo.	8.0	14.6	3.70	42.5	0.8		114.8	39.4	34.3	17	28	101	7.3
11. K	67	M	10 mo.	12.1	14.3	5.38	46	0.8	7.8	86	27	31	19			6.4
12. L	63	M	14 mo.	15.3	15.7	4.37	43.5	0.5	7.4	99	33	36	15	24	98	6.2
13. M	74	M	17 mo.	14.7	13.3								14			5.9
14. N	66	M	22 mo.	9.2	11.4		34						19	27	104	4.2
15. O	83	M	24 mo.	11.5	13.3	3.25	42	0.9	8.0							
16. P	79	M	26 mo.	8.9	12.5	3.51	37	0.3	7.1	105	35	33.8	33			7.1
17. Q	69	M	31 mo.	14.0	12.3	3.35	35	0.8	8.8	106	36.7	34.6	19	26	105	5.9
18. R	68	M	36 mo.	13.1	12.6	3.35	39	1.2					13	25	107	6.0
19. S	68	M	41 mo.		7.2	1.92	22.3	2.3	9.4	116	37.5	32.7	11			6.0
20. T	28	F	6 yr., 2 mo.		13.9	3.94	43.4	0.7	7.4	110.2	35.2	32	10			6.8
21. U	70	M	6 yr., 4 mo.	14.7	14.7	3.55	42.4	1.2					17	26	108	5.9
22. V*	68	M	8 yr., 9 mo.	10.7	14.3	3.2	45.0	1.2	8.4	140.6	44.7	31.8	22	28	105	7.3
							Near-total Gastrectomy									
23. W	72	M	2 yr.	12.3	14.5	4.86	44	0.5	6.8	90.3	29	31.6	25			6.2
24. X	46	M	4½ yr.	16.3	18.5	5.06	50	0.8	6.8	98.8	36.6	37	21			7.1
25. Y	66	M	6 yr.	11.5	13.1	3.78		1.1	7.6				12			6.4
26. Z	65	M	6 yr.	15.0	10.1	2.43							18	26	109	5.8

*Treated blood abnormality.

tric polyps. She subsequently had a total gastrectomy because of residual polyps with continued bleeding. The sixth patient (Case 17) showed only macrocytosis. He had been taking liver injections regularly, although no definite history of anemia could be obtained.

Three additional cases showed macrocytosis as the only abnormality.

Three other patients were anemic at the time of this study. One of these (Case 19) was a typical megaloblastic anemia occurring three years and five months after a total gastrectomy for carcinoma. The other two were hypochromic anemias.

All cases of megaloblastic anemia responded satisfactorily to the usual methods of treatment.

Therefore, excluding the one case in which it was known that a blood abnormality was present prior to gastrectomy, eleven of twenty-six patients showed some type of blood abnormality when examined at varying intervals following total or near-total gastrectomy. Five of these abnormalities were megaloblastic anemias occurring at intervals of one and one-fourth, three and one-fourth, five, five, and six years following operation, respectively. Two of the abnormalities were mild hypochromic anemia, and the remaining four abnormalities consisted of macrocytosis. Of eleven patients alive two years or longer after gastrectomy, five developed a megaloblastic anemia.

Blood urea nitrogen, CO_2, chlorides, and total serum protein showed no consistent deviation from normal.

6. *Iron tolerance test:* None of the patients studied showed fasting serum iron levels significantly altered from normal. Six of eleven patients showed strikingly little change in the serum iron level after the oral administration of 1 Gm. of ferrous gluconate.

TABLE VI. IRON ABSORPTION AFTER TOTAL AND NEAR-TOTAL GASTRECTOMY

PATIENT	POSTOPERATIVE INTERVAL	SERUM IRON* (γ/100 C.C.)			
		FASTING	1 HR.	3 HR.	5 HR.
1. A	4 mo.	75	67	77	72
2. C	4 mo.	54	98	103	67
3. E	5 mo.	62	118	100	
4. F	7 mo.	145	125	180	135
5. G	8 mo.	65	65	65	68
6. K	22 mo.	107			
7. O	41 mo.	192	195	170	166
8. P	5 yr., 2 mo.	110	140	115	65
9. F. H.†	2 yr.	52	56	66	83
1. S	2 yr.	122	149	100	392
2. T	4½ yr.	138	150	125	107
3. V	6 yr.	176		202	213

*1.0 Gm. ferrous gluconate given orally after fasting specimen taken.
†Not included in other tables.

7. *Roentgenologic studies:* Seven of seventeen patients showed some narrowing at the site of anastomosis of the esophagus to the intestines. Six of these were symptomatic, but only two were severe enough to interfere with

alimentation seriously. One of these was successfully dilated with restoration of normal swallowing. The other patient was uncooperative and refused further dilatation after one failure due to technical difficulties.

TABLE VII. X-RAY STUDIES IN CASES OF TOTAL AND NEAR-TOTAL GASTRECTOMY

PATIENT	AGE (YR.)	SEX	POSTOPERATIVE INTERVAL	NARROWING ANASTOMOSIS (0-4 + SCALE)	SMALL BOWEL PATTERN	PASSAGE TIME TO CECUM
				Total		
1. A	69	M	4 mo.	+++	N	4 hr.
2. C	70	F	4 mo.	0	Enlargement and coarsening of folds in jejunum	7 hr.
3. E	28	F	5 mo.	+	N	
4. F	75	M	5 mo.	++	Decreased motility retention of Ba in upper jejunum 2 hr. after ingestion	
5. H	46	F	8 mo.	+	N	1⅔ hr.
6. I	60	M	8 mo.	0	Small bowel empties rapidly	
7. J	71	M	9 mo.	0	N	4 hr.
8. K	67	M	10 mo.	0	N	2 hr.
9. L	63	M	14 mo.	+	N	4 hr.
10. N	79	M	22 mo.	0	N	4 hr.
11. O	83	M	24 mo.	0	N	½ hr.
12. P	66	M	14 mo.	+++	Spastic areas	
13. Q	69	M	31 mo.	0	N	7 hr.
14. R	68	M	36 mo.	+	N	
15. T	28	F	5 yr.	0	N	
16. U	70	M	6 yr., 9 mo.	0	N	3 hr.
17. V	68	M	8 yr., 9 mo.	0	Moderate dilatation of proximal jejunum with moderate delay in emptying	
				Near-total		
1. W	72	M	2 yr.	0	N	Gastric pouch emptied 2 hr.
2. X	46	M	4½ yr.	0	N	Gastric pouch emptied 1 hr., transit to colon 3 hr.
3. Z	65	M	6 yr.	0	N	Pouch empties with extreme rapidity; 5-6 hr. for Ba to reach colon

Three cases showed some dilatation and stasis in the upper jejunum. The total transit time through the small intestine was in the normal range in nine of the twelve cases in which it was determined.

8. *Esophagitis:* Eleven patients were examined by esophagoscopy. Two of these showed a mild esophagitis in the distal esophagus as evidenced by increased reddening and tendency to bleed on contact. One patient had an esophagoduodenal anastomosis and the other a Roux Y type of reconstruction. A third patient showed a moderate esophagitis as evidenced by more pronounced redness of the mucosa. This patient had an esophagojejunal anastomosis with enteroanastomosis. The interval since operation was ten months,

seven months, and two months, respectively. A third patient wtih an esophagojejunostomy and enteroanastomosis showed a questionable esophagitis when examined forty-one months following operation.

TABLE VIII. ESOPHAGITIS AFTER TOTAL AND NEAR-TOTAL GASTRECTOMY

PATIENT	POSTOPERATIVE INTERVAL	TYPE OF ANASTOMOSIS	ESOPHAGITIS
1. G	7 mo.	Roux Y	Mild
2. H	8 mo.	Roux Y	None
3. K	10 mo.	Esophagoduodenostomy	Mild
4. M	17 mo.	Roux Y	None
5. O	24 mo.	Roux Y	None
6. S	41 mo.	Esophagojejunostomy	Questionable
7. T	2 mo.	Esophagojejunostomy	None
8. U	6 yr., 4 mo.	Esophagojejunostomy	None
9. V	8 yr., 9 mo.	Esophagojejunostomy	None
10. TQ*	6 mo.	Esophagojejunostomy	None
11. AB*	3 mo.	Esophagojejunostomy	Moderate

*Not included in other tables.

DISCUSSION

Disturbances in nutrition and hemopoiesis are the main categories of undesirable side effects which may occur following gastrectomy.

Disturbances in Nutrition.—Weight loss and weakness are the usual manifestations of nutritional insufficiency. In the cases under consideration in this report, weight loss was moderate to marked with apparently little improvement with the passage of time. Many reports[3, 13-16] indicate that there is a gain in weight following partial or total gastrectomy. These figures are of less importance in assessing the nutritional status of the patient than are figures which compare the postoperative weight to the normal preoperative weight. Wells and Welbourn[17] reported that patients treated by total gastrectomy showed an average of fourteen pounds' weight loss before operation and another sixteen pounds' after operation. Brain and Stammers[18] stated that after total gastrectomy patients look like the inmates of a concentration camp. Everson,[19] in a collective review, noted that only three of forty patients treated by total gastrectomy regained normal preoperative weight. The remainder were below normal weight by an average of twenty-six pounds. In the experimental animal, total gastrectomy is frequently followed by moderate to severe weight loss.[20] Ivy and associates,[23] however, reported that if dogs are fed about twice the normal amount after total gastrectomy they will remain in good nutrition.

In many patients, the so-called normal preoperative weight represents moderate to severe obesity, so that it might be argued that the reduction in weight after total gastrectomy still leaves the patient in the normal range for his age and height. Using the Metropolitan Life Insurance Company tables for average weight correlated to height, body build, and sex, we find that three of nine patients with total gastrectomy fall in the normal range given. The remaining six fall below average optimum weight by an average of 12 per cent (median = 12 per cent). However, two-thirds of the patitents in this report complained of weakness, suggesting the likelihood of true impairment of nutrition.

In general, the major factors involved in weight loss must be (1) inadequate intake, (2) inadequate assimilation of the ingested food, or (3) a combination of the two factors.

1. *Inadequate intake:* The obvious factors involved here are (a) anorexia or (b) pain, a sense of fullness or discomfort or disagreeable sensations commonly termed the "dumping syndrome" which may occur after the ingestion of a relatively small amount of food. The dumping syndrome has been well described after partial gastrectomy. Most investigators[16, 17, 39, 40, 42, 43] agree that the sudden filling of some portion of the upper gastrointestinal tract with food is responsible for the early symptoms. Gholiger[63] reported that 100 per cent of patients with total gastrectomy have the dumping syndrome. The impression gained from careful questioning of the patients in this study is that dietary intake is generally reduced moderately to markedly when compared to the normal preoperative quantity. Furthermore, nearly one-half of the patients regularly experienced distress or pain following meals which, although generally mild, undoubtedly results in a reduction of food intake.

2. *Inadequate assimilation:* Much importance has been attached to the impaired assimilation of dietary fat as an etiological factor in poor nutrition after total gastrectomy. Everson[19] reported impaired fat assimilation in twenty-one of thirty-three collected patients, and impaired protein assimilation in six of fourteen patients. Of fifteen dogs studied, all showed increased fecal fat excretion, and fifteen of sixteen reported dog studies showed increased protein loss in the stool. Brázda[15] reported the excretion of 12 to 37 per cent of the ingested fat (107 Gm. per day) in ten patients with total gastrectomy with a mean value of 28.5 per cent. Our own figures of 5.8 to 20 per cent fat excretion with a mean of 11.5 per cent in fourteen patients represents only a mild abnormality in assimilation of fat (diet of 80 Gm. per day). Protein assimilation was normal. There was no correlation with the interval since operation. The commonly suggested etiological factors in the impaired assimilation of dietary fat following gastrectomy are (a) deficient pancreatic secretion secondary to inadequate stimuli, vagotomy, and absence of sufficient hydrochloric acid to activate the secretin mechanism, (b) inadequate mixing of the food with the biliary and pancreatic secretions, and (c) increased rate of transit of food through the small intestine.

The role of bile and pancreatic juice in the digestion and absorption of fat is well known. Annis and Hallenbeck[24] noted in dogs after partial gastrectomy that the volume, total bicarbonate, total nitrogen, and trypsin activity of the pancreatic juice were greatly reduced. Howard and co-workers[25] found, in studying a patient with a pancreatic fistula following a bullet wound, that a test meal introduced by intestinal tube into the jejunum produced only slight stimulation of pancreatic secretion, whereas introduction of the same meal into the duodenum caused a much greater flow of juice. A maximal response was obtained when the meal was eaten. This suggests the importance of the passage of food through the duodenum in eliciting pancreatic secretion. Vagotomy in itself is said by some authors,[26, 27] to decrease pancreatic secretion, but a more recent work favors the opinion that no significant altera-

tion occurs.[28, 29] Shingleton[30] reported that after vagotomy the fasting volume of duodenal content is increased but the pancreatic enzyme content is decreased. The impression obtained from the study of pancreatic secretion after total gastrectomy in the present paper is that the pancreas is capable of normal secretion.

Inadequate stimulation and poor mixing secondary to by-passing the duodenum, delayed secretory response, and increased rate of passage of the ingested food are probably of greater importance in the etiology of impaired fat and protein assimilation than any primary decrease in pancreatic secretion.

Wollaeger[31] reported the mean fecal fat excretion to be 8.7 Gm. per day in the normal individual, 12.3 Gm. per day after the Billroth I type of gastrectomy, and 19.9 Gm. per day after the Billroth II gastrectomy when the patient is on a high fat diet (200 Gm. per day). The difference was found to be statistically significant, and he concluded that Billroth I gastrectomy is followed by less gastrointestinal disturbance than Billroth II. Bohmansson[32] came to the same conclusion, which he attributed to better mixing with bile and pancreatic juice when food traversed the duodenum plus more normal emptying after Billroth I. Everson[33] found that dogs excreted more of the dietary nitrogen after total gastrectomy with esophagojejunal anastomosis than with esophagoduodenal anastomosis. Calabi and associates[34] studied a patient with an esophagodermojejunoplasty, who also had a gastrostomy because of esophageal stenosis. They found that fat assimilation was normal when the diet was given by gastrostomy but abnormally decreased when fed orally, by-passing the stomach and duodenum. They attributed the difference to the storage function of the stomach and the gradual emptying in small prepared amounts of food from the stomach. Dragstedt[35] showed in the dog that removal of 80 to 90 per cent of the pancreas did not alter the normal assimilation of fat and protein. However, ligation of the pancreatic ducts or pancreatectomy causes excessive excretion of fat and protein in the stool, approximately 60 per cent of the fat and 40 per cent of the protein ingested being lost in this way. Similarly, diversion of all bile from the upper intestinal tract in dogs causes them to excrete quite constantly 60 per cent of their dietary fat.[36, 37] Large doses of ox bile extracts or bile salts were necessary for complete fat absorption. Since vagal control and hormonal regulation from the duodenum are known physiologic mechanisms in biliary tract emptying, a situation exists parallel to the pancreas, which can help explain alterations in the assimilation of dietary fat after gastrectomy. Here again inadequate stimuli, delayed response, and poor mixing appear to be likely factors.

As mentioned earlier, changes in the rate of passage of food through the gut may cause poor mixing with the digestive juices and decreased absorption of the ingested diet. Freeman[21] showed in monkeys following total gastrectomy that barium passed rapidly through the upper small intestine but underwent compensatory slowing in the lower ileum. Bruusgaard[41] found in human beings after partial gastrectomy for peptic ulcer that evacuation of the small intestine is accelerated in the proximal 30 to 50 cm. while passage through the whole small intestine is little changed from normal. Wells and Welbourn,[17] who studied 300 cases after gastrectomy, asserted that the rapid entrance of

a large bulk of food into the jejunum stimulates active peristalsis, which transports the food down the small bowel very rapidly. In many cases, the increased activity affects the jejunum only and there is a compensatory slowing in the ileum. After total gastrectomy this effect is exaggerated. Farris, Ransom, and Coller[14] noted that all gastric operations may be followed by transient increased motility. Ingested powdered charcoal may appear on the stool in twelve hours. They cited two long-term survivors after total gastrectomy in whom barium studies show, if anything, slightly decreased motility. Two of twenty of our patients showed this delayed emptying with puddling in the jejunum, two patients showed rapid emptying of the entire small bowel, and the remainder were in the normal range. Weigen and co-workers[44] found no consistent change in gastric emptying or intestinal transit by means of roentgenologic studies after total pancreatectomy in dogs. Alvarez[45] noted hyperirritability of the small bowel as a constant finding after vagotomy in rabbits. To summarize, there is evidence suggesting that after gastrectomy there is alteration in gastrointestinal motility, particularly in the upper small bowel, with quickened transit. The importance of this in the etiology of assimilation disturbances is apparent and has been discussed previously.

Disturbances in Hemopoiesis.—Following the epic work of Minot and Castle in elucidating much of the problem of pernicious anemia, a great deal of attention was given to experimental work in an effort to produce this disease in animals. Petri and Jensenius[46, 47] and Jacobsen and Plum[48] reviewed much of this work and concluded that gastrectomy alone or combined with extirpation of various segments of the intestinal tract has not been successful in producing the typical picture of pernicious anemia. Gastrectomy in dogs, swine, cats, monkeys, and rats is followed frequently by a more or less pronounced anemia, usually hypochromic in variety. Other features seen are inhibition of growth, emaciation, changes in the skin and hair, and sometimes disturbances in bone calcification. Several authors[48] have shown that extracts from the liver of gastrectomized animals have no therapeutic effect in human pernicious anemia. This suggests, of course, that storage of antipernicious anemia principle is greatly reduced, but the fact that a typical anemia does not occur in the animal would seem to indicate that some absorption is still taking place.

Numerous reports have been made on the appearance of megaloblastic anemia occurring after total gastrectomy and also partial gastrectomy and gastroenterostomy.[2, 19, 49-53] In the main, this anemia closely resembles pernicious anemia. Beebe and Meneeley,[54] in reporting such a case, surveyed the literature and found fifty-five reported cases of pernicious anemia following total or partial gastrectomy. These occurred from two to five years after operation.

MacDonald,[55] in 1947, reviewed the literature on forty-six patients surviving total gastrectomy by three years or more. In twelve of these cases the blood findings resembled pernicious anemia and in five others the findings were doubtful. Of the remainder, many had been receiving liver therapy, although no definite history of anemia could be determined. Macrocytosis has

been noted as the only abnormality after total gastrectomy; Brain and Stammers[18] reported this condition in eleven of forty patients following total or near-total gastrectomy. Schwartz and Legere[56] studied fifty-four patients with pernicious anemia, and found that following complete cessation of liver therapy it took from two to thirty-eight months for hematologic relapse to occur. Landboe-Christensen[57] recently found evidence suggesting that the human duodenum may contain a small amount of intrinsic factor. If this is proved, it would help to explain the long interval apparently necessary before megaloblastic anemia occurs after total gastrectomy in the human being. Since comparatively few patients who have required total gastrectomy have survived for the requisite length of time, it is not too surprising that megaloblastic anemia is encountered relatively infrequently. However, the likelihood of megaloblastic anemia developing after total gastrectomy need have no influence on the decision to employ this procedure, as the anemia is readily managed by the same methods used in the treatment of pernicious anemia.

Hypochromic microcytic anemia is more commonly encountered after gastrectomy. Many reports have been made, with most figures ranging from 10 to 20 per cent. Women have a higher incidence, and when considered separately are seen to have from two to three times as many anemias. Bruusgaard[41] found that after partial gastrectomy 44 per cent of the women and 28 per cent of the men were anemic. The difference in these figures is accounted for by the higher incidence in the menstruating female, the women under 40 showing an incidence of 59 per cent, whereas those over 40 had an incidence of 28 per cent, the same as for men.

Lyngar[58] found that the serum iron was low in patients with hypochromic anemia after gastrectomy. Iron absorption studies showed poor absorption in some cases. All patients responded eventually to iron therapy, although some finally required intravenous administration.

The absence of HCl, which frees combined iron and reduces it to the ferrous state necessary for absorption, the by-passing of the duodenum in many cases, which is the most active site in iron absorption, and increased motility through the upper intestines are all factors which can explain inadequate iron absorption.

Starkenstein[59] found that maximal utilization of iron complexes was obtained when they were administered per os with the addition of fats. Vannotti and Delachaux[60] suggested that this may indicate that the duodenal absorption of iron is effected with the help of bile. Disturbances in regard to the secretion of the latter have been mentioned earlier.

In our series of cases, megaloblastic anemia has been more frequent than hypochromic.

To recapitulate briefly, after total or near-total gastrectomy, megaloblastic anemia may be expected to occur frequently when patients survive for a long enough period of time. This anemia is apparently the same as the spontaneously occurring pernicious anemia, and responds to the same management. Hypochromic microcytic anemia is also seen and is apparently secondary to inadequate iron absorption. Treatment with iron orally or parenterally is uniformly successful.

Iron absorption as seen by the iron tolerance test is suggestively impaired in six of eleven of the patients studied in the group reported herein.

Recent studies by Ferguson and associates[61] and Cross and Wangensteen[62] have shown the great sensitivity of the esophageal mucosa to gastric juice and, to a lesser extent, to bile and pancreatic juice. That regurgitation of bile and pancreatic juice occurs after total gastrectomy is known. Regurgitation would probably be most likely after esophagoduodenostomy and less so after esophagojejunostomy with esophagoenterostomy or the Roux Y procedure. While esophagitis has been little noted in the literature, we found mild to moderate esophagitis in three of eleven cases, plus one questionable case. None of these patients had symptoms, however. The importance of this observation remains to be determined.

SUMMARY AND CONCLUSION

Twenty-two patients with total gastrectomy and four patients with near-total gastrectomy were studied at varying intervals of two months to eight years, nine months after operation. Information was obtained in regard to weight loss, eating habits, gastrointestinal symptoms, roentgenologic study of the upper gastrointestinal tract, pancreatic function, fecal fat and nitrogen, iron absorption, hematology, and esophagitis. This information is tabulated in detail and discussed in brief form. The present study indicates that total gastrectomy is regularly followed by loss of weight and, not uncommonly, weakness of a mild degree. While loss of dietary fat in the feces is somewhat increased, on the average the amount involved is not sufficient to account for the degree of weight loss. The more likely explanation is reduced food intake secondary to loss of the storage function of the stomach and the occurrence of distress after eating more than a small amount of food at one time.

Anemia may be expected to occur frequently, particularly if the patient survives for a long enough period of time after operation. This anemia presents no particular problem in management and should not influence the decision to use the procedure. Dietary regulation using a high-caloric, high-protein, high-fat diet with small frequent meals would appear to offer the most logical approach to management. Supplemental medication, such as pancreatin, Tween 80, atropine, and so forth, has been recommended by various authors and may be worthy of trial in difficult cases.

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